

**ANALYTICAL STUDY OF PROGNOSTIC FACTORS
AND OUTCOME IN TRAUMATIC BRAIN INJURY IN
PATIENTS AGED 18 TO 40 YEARS**

**DISSERTATION SUBMITTED FOR
M.Ch., BRANCH II (NEUROSURGERY)
DEGREE EXAMINATION**



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CERTIFICATE

This is to certify that this dissertation entitled “**ANALYTICAL STUDY OF PROGNOSTIC FACTORS AND OUTCOME IN TRAUMATIC BRAIN INJURY IN PATIENTS AGED 18 TO 40 YEARS**” submitted by **Dr.H.M.BABA DHOULATH KHAN** to The Tamil Nadu Dr. M. G. R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.Ch.,(Neurosurgery) and is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, **Dr.H.M.BABA DHOULATH KHAN** solemnly declare that I carried out this work on **“ANALYTICAL STUDY OF PROGNOSTIC FACTORS AND OUTCOME IN TRAUMATIC BRAIN INJURY IN PATIENTS AGED 18 TO 40 YEARS”** at Department of Neurosurgery, Government Rajaji Hospital, Madurai during the period of Jan 2010 –Dec 2011.

I also declare this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to the TamilNadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M.Ch., (Neurosurgery) Degree examination.

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INTRODUCTION

Care of head injured patients forms an important part of neurosurgeon's work in all countries and especially in the developing countries, where widespread facilities for such care may be meager. The incidence of head injury is increasing globally every year with the advent of increasing technological modernization, industrialization and urbanization. According to WHO, about 3.5 million people die all over the world due to multiple injuries as well as head injuries, road traffic accident being the most common cause. In our country also, there is an ever increasing number of road accidents and we have the dubious distinction of having the highest incidence of such accidents per 1000 vehicles or deaths per 1000 accidents. More over in many occasions, the post traumatic sequelae including the intellectual dysfunction and residual deficits result in huge socio economic burden to both the family and the country.

The speed of modernization of roads and establishment of roads of international standard does not cope up with the increasing number of vehicles, which adds to the problem, especially in semi-urban and rural areas of the country. The attitude and ignorance of the public including poor awareness of traffic rules, not wearing safety helmets, drunken driving, use of mobile phones while driving, etc. also play a vital role in causing increasing number of road accidents in all age groups.

Although, well organized head injury services are increasing year by year in both public sector and private sector, yet inadequate to meet the disproportionately increasing demand due to accidents and head injuries.

Availability of first aid, skilled attention at the site of injury, improved diagnostic techniques, better resuscitation methods and clearer understanding of metabolic and bio-chemical aspects will help to reduce the morbidity and mortality in a seriously head injured patients. In recent years, there is a marked change in the evaluation & management of head injury and therefore there is a marked reduction in the number of deaths

in injury patients with a proportionate increase in the number of dependent patients.

With the development of modern Neuro Surgical Intensive care units with 24 hours monitoring of the patients neurological state, early intubation tracheostomy and ventilator care that can be continued for several days (or) weeks as well as advanced 64 slice C.T., have made an enormous impact on the management of head injuries in the recent years.

In general, Head injury plays a significant cause for death among patients those who die of multiple injuries. All over the world, the majority of head injury victims of traffic accidents belong to the young and productive age group. Very often being the major earning members of the family the impact on young adults is manifold which cripples not only the individual but also the family and the nation.

HISTORICAL BACKGROUND

Skulls of the Neolithic period show evidence of fractures and man made defects and bear mute testimony to the fact that one of the earliest forms of surgery to be practiced by man was for head injuries. Signs of bony proliferation around such defects also indicate that the patients often survived for considerable periods after the injury and surgery.

The Incas of Peru probably practiced trephination as far back as 3000 B.C. The Edwin Smith Papyrus, recording the surgical practices in Ancient Egypt (1700 B.C.) recognized that scalp lacerations and fractures of the skull with meningeal irritation could be treated. The combination of the pulsating brain in the wound, bleeding from both nostrils and stiffness of the neck were recognized to be of grave prognostic significance and the surgeon was cautioned that this was ailment not to be treated.

At the time of Hippocrates (460-370 B.C.) different types of fractures were recognized and trephination was advocated. Extradural

hemorrhage without skull fracture as well as intradural hemorrhage were known in ancient Greece and Rome. One of the earliest cases of subdural hemorrhage on record was that of Henry the Second of France who sustained a frontal wound and died. Hoessly published a translation of Wepfer's case notes written in 1657 on subdural haematoma. Ambrose Pare in the seventeenth century recognized concussion as a distinct clinical entity.

Hutchinson, in 1867, drew attention to the significance of the unilateral dilated pupil in head injury. Cushing, in 1908, advocated subtemporal decompressive operations for the intracranial complications associated with fractures of the skull.

Evidence of the of the practice cranioplasty is available in five thousand year old Peruvian skulls. Gourd, shell, bone, coconut, silver, gold and lead have been tried for this purpose, down the centuries. Aluminium, tantalum, vitallium, platinum, titanium and stainless steel have been tried recently. Zander is reported to have been the first to perform, in 1940, methyl methacrylate cranioplasty in the human.

The importance of prevention of head injuries has been recognized for a long time, since the knights of the middle ages wore steel helmets as part of their armour. The tin hat was evolved during the First World War and the crash helmet of the Second World War was its natural extension. To Cairns goes the credit for popularizing the crash helmet for civilian use.

REVIEW OF LITERATURE

A wealth of literature has focused on the associations between predictors and outcome in univariate analysis. Most studies have concentrated on patients with severe and moderate TBI. Much information on the univariate association between predictors and outcome is contained in the section “Early Indicators of Prognosis in Severe Traumatic Brain Injury” of the Brain Trauma Foundation’s Guidelines on Management and Prognosis of Severe Head Injury, first published in July 2000. A series of papers reported details correlating the GOS and demographic characteristics³¹, cause of injury⁸, GCS and pupil response²⁵, secondary insults²⁶, blood pressure⁹, computed tomography (CT) scan features²¹, and laboratory parameters³⁹.

Among the studies that included multivariable analysis, two systematic reviews on prognostic modeling, the CRASH model and the IMPACT model have shown the shortcomings of many of the studies that reported on prognostic models previously. The IMPACT study group³⁶ reported the results of extensive prognostic analyses performed

in a meta-analysis of individual patient data from eight randomized controlled trials and three observational series that included more than 9000 patients.²⁴ The CRASH model presented by the MRC CRASH trial collaborators has included patients' data from low and middle income countries also. The results of multivariable analyses reporting also on the added predictive value were presented in the same series by Murray and colleagues³⁰.

Conceptually, the main predictors of outcome after TBI can be grouped together into “building blocks”, some of which are modifiable and some not. Current knowledge on these “building blocks” and parameters is summarized in the following sections.

Genetic Constitution

In this era of discovery of the human genome, several genes and their polymorphisms are under investigation in patients with TBI. The presence of the apolipoprotein E4 allele is associated with poorer functional recovery¹. Other genes for which evidence exists for an association with poorer outcome are P53, COMT, DND2 and CACNAIA¹⁷.

Demographic Factors

Age is the strongest and most extensively studied predictor of outcome after TBI. Many publications on prognostic effects exist, all stating that older age is correlated with poorer outcome. It is remarkable that most studies have analyzed the association between age and outcome with threshold values. Different thresholds varying from 30 to 60 years of age have been used. Studies using higher threshold levels report poorer outcome in the upper age group, and a morality rate of greater than 75% has been described in patients older than 60 years with severe TBI^{32, 20, 5}. A continuous age dependency was described in only few publications in the past^{11,15}.

In a recent meta-analytic study of individual data from more than 9000 patients, a continuous effect of age on outcome was described that could be approximated by a linear function. Threshold values could not be identified. Other demographic factors studied for their association with outcome include gender, race and education.

Males are more prone to sustain a TBI because of higher risk for road traffic accidents and assaults, but a clear association between

gender and outcome assessed by the GOS has not been shown for TBI. This, however, does not exclude the possibility that there may be some effect of gender on outcome. In a meta-analysis Farace and Alves found poorer outcome in females surviving severe TBI than in males¹². Experimental studies, however, indicate that progesterone may have a neuroprotective effect, and consequently, prognosis in females may be better if TBI is sustained at a time of the menstrual cycle when progesterone levels are high.

The possible association between race and outcome after TBI has not been extensively studied. Two smaller studies showed poorer outcome in black patients^{18,40}, but other did not find a clear association^{35,7}. The IMPACT study group, in contrast, studying data from 5320 patients, found a statistically significant association between race and outcome, with black patients having a poorer outcome³¹. It was hypothesized that this might be due to difference in severity or cause of injury. Such, however, proved not to be the case, and after adjustment for cause of injury, age, motor score, and pupils, the prognostic effect was even stronger.

These data are convincing and leave little room for doubt, but what the underlying reason for this association is can only be speculated. The response to injury may be different because of genetic constitution and biologic differences, or possible access to acute and postacute care may be more limited.

A weak association between level of education and outcome has been reported^{31,40}. Mushkudiani and coworkers, however, clearly showed that this weak association could be explained by other factors and disappeared on adjustment.

Clinical Severity

Clinical severity is an important prognostic factor that can be assessed in all patients. Severity in patients with TBI is related to both extracranial and intracranial injuries. The overall severity of extracranial injuries is commonly assessed with the Abbreviated Injury Score (AIS)² or the Injury Severity Score (ISS)³. Although an adverse effect of the presence of extracranial injuries on outcome after TBI is well documented²⁷, the prognostic value of overall injury severity and extracranial injuries has not been well documented in the literature.

Walder and associates found a stronger association between the AIS and outcome than assessment of the GCS alone ⁴¹.

The clinical severity of intracranial injuries is reflected by the level of consciousness as assessed by the GCS³⁸. Many studies have demonstrated an association between lower scores on the GCS and poorer outcome. In patients with more severe injuries, the motor component of the GCS has the greatest predictive value because eye and verbal responses are commonly absent in these patients. It should be recognized that GCS scores may fluctuate early after injury, with some patients deteriorating and others improving. From a perspective of prognosis, assessment of the GCS should therefore be related to a given time period, depending on the intent for estimating prognosis. For purpose of classification and summarizing baseline characteristics before in-hospital therapeutic interventions, the GCS is commonly assessed on admission after primary respiratory and hemodynamic stabilization. Reliable assessment of the GCS, however, may be obscured in the acute setting by confounders such as medical sedation, paralysis, or intoxication ^{25,4,37}. Marmarou and coauthors reported a stronger

association with outcome for an abnormal absent motor response than for an absent motor response²⁵. The most likely explanation for this is that the category of patients scored as having an absent motor reaction will include “false-absent” scores because of the confounding effects of sedation and paralysis.

Abnormalities in pupillary reactivity reflect brainstem compression and are strongly associated with poorer outcome²⁴. Marmarou and colleagues reported that pupillary reactivity was a more stable parameter in the early phase after injury than the GCS score because it is less prone to the influences of sedation and paralysis.

Secondary Insults

The injured brain is more vulnerable than a normal, healthy brain to systemic secondary insults such as hypoxia and hypotension. In the experimental but also in the clinical situation, the occurrence of secondary insults increases the degree of secondary damage after injury. The presence of secondary insults is associated with poorer outcome^{29,42} and the depth, duration, and number of hypotensive insults all cumulate toward poorer outcome^{26,23}. Most studies have focused on early

hypotensive and hypoxic events in which hypotension was defined as any episode with a systolic blood pressure lower than 90mm Hg. The association between the actual blood pressure on admission and outcome has been further analyzed in a continuous manner by the IMPACT study group³⁰. These studies, which incorporated data from 6801 patients, showed that the relationship between blood pressure and outcome in continuous low blood pressure and high blood pressure are both associated with a poorer outcome. After adjustment for age, motor score, and pupillary reactivity, the effects of higher blood pressure, however, largely disappeared thus indicating that this association is most likely secondary to increasing severity of the injury. Various studies have shown that combination of hypoxia and hypotension has a greater adverse effect on outcome than can be explained by either insult alone, the effects; however, appear to be sub additive rather than the synergistic.

Structural Abnormalities

CT is the investigation of choice in the acute phase after TBI to identify the presence and extent of structural damage. The relevance of

CT scanning for the purpose of classification and prediction has increased with the growing difficulties in reliable assessment of clinical severity according to the GCS ^{4,6,28}. The prognostic value of individual CT characteristics in patients with TBI including status of the basal cisterns, midline shift, the presence and type of intracranial lesions and traumatic subarachnoid hemorrhage (tSAH) is well documented. In 1991 Marshall and associates introduced a descriptive system of CT classification that focuses on the presence or absence of a mass lesion and differentiates diffuse injuries by signs of increased intracranial pressure (ICP) such as compression of the basal cisterns and midline shift.

This classification is also strongly related to outcome with the poorest prognosis in patients with CT category IV (signs of raised ICP plus shift) and the best outcome in patients without visible structural abnormalities. The Marshall CT classification, however, has limitations, such as the broad differentiation between diffuse injuries and mass lesions and the lack of specification of the type of mass lesion. Thus, this classification might mask signs of raised ICP in addition to a mass

lesion and does not fully use the prognostic information contained in the individual CT characteristics scored. Mass and coworkers proposed a score chart for assessing the risk for poorer outcome based on individual CT characteristics and showed that such a chart results in better discrimination between patients with better versus poorer outcome than does the descriptive Marshall classification²². This advantage was confirmed in subsequent work by Flint and colleagues¹³. The prognostic relevance of tSAH was extensively described by Kakariieka and associates after extensive analysis of the nimodipine studies. Later work confirmed the presence of tSAH as one of the strongest CT predictors of outcome after TBI¹⁹. Most studies, however, have concentrated on the presence or absence of tSAH without differentiation of the location (basal cisterns versus cortical) or extent. Cortical tSAH is frequently associated with underlying contusions, and its relevance is probably different from that of tSAH in the basal cisterns, which may incur an increased risk for vasospasm.

Laboratory Parameters

Relative few studies have investigated the relationship between laboratory parameters on admission and final outcome. Abnormal values may also be modifiable. Various studies have shown a strong relationship between a poorer outcome and higher glucose values, low hemoglobin, low platelets, and coagulation disturbances. The results from the IMPACT studies have demonstrated that the addition of the laboratory values to a prognostic model increases discrimination. These studies showed the greatest discriminatory properties for coagulation abnormalities and glucose. Although laboratory values may be modifiable, the observed association between abnormal values and poorer outcome, however, does not by definition mean that correcting these abnormal values will indeed improve outcome. The observed abnormality may simply be an expression or surrogate marker of the severity of injury. Currently, there is great interest in various biomarkers released from damaged or necrotic neurons and glial cells in the brain. Various experimental and preliminary clinical studies have confirmed this potential.

Multivariate Prediction Models: Single predictors often have insufficient predictive value to distinguish patients who will do well from those who will do poorly. Moreover, patients can have different characteristics that affect the prognosis in opposite directions. For example, for a 24-year –old patient with fixed pupils, we would predict a favourable outcome based on age but an unfavourable outcome based on pupil reactivity. Thus, estimation in prediction research is by definition a multivariable challenge in which multiple risk factors need to be considered jointly with multivariable analysis. For this purpose, relevant prognostic factors are combined in a prediction model and often presented as rules or nomograms.

Many international multivariate prediction models are available for the study of prognostic factors and their significance in outcome. Recently, two prediction models, developed from large patient series and externally validated, have been published: models presented by the MRC CRASH trial collaboration and a prediction model proposed by the IMPACT study group. The CRASH model also included patient

data from low and middle income countries. Importantly, both models were developed from data available on admission, before provision of specialist care. They are therefore ideally suited for a baseline calculation of prognostic risk. Both models showed good performance in terms of both discrimination and calibration. Both approaches confirmed that the largest amount of prognostic information was contained in a core set of three predictors: age, motor score, and pupillary reactivity. The IMPACT study group further evaluated the additional benefit of adding more “building blocks”, such as structural imaging (CT characteristics), secondary insults, and laboratory data. Better performance was noted in a model that included this information.

CRASH MODEL

This prognostic model may be used as an aid to estimate mortality at 14 days and death and severe disability at six months in patients with traumatic brain injury (TBI). The predictions are based on the average outcome in adult patients with Glasgow coma score (GCS) of 14 or less, within 8 hours of injury, and can only support -

not replace - clinical judgment. Although individual names of countries can be selected in the model, the estimates are based on two alternative sets of models (high income countries or low & middle income countries). The CRASH model proforma includes the following parameters: country, age, GCS, pupillary reaction, presence or absence of extra cranial injuries and CT scan.

IMPACT MODEL :

Based on extensive prognostic analysis the IMPACT investigators have developed prognostic models for predicting 6 month outcome in adult patients with moderate to severe head injury (Glasgow Coma Scale ≤ 12) on admission. By entering the characteristics into the calculator, the models will provide an estimate of the expected outcome at 6 months. There are three models of increasing complexity for predicting outcome (Core, Core + CT, Core + CT + Lab). These models were developed and validated in collaboration with the CRASH trial collaborators on large numbers of individual patient data (the IMPACT database). The models discriminate well, and are particularly suited for

purposes of classification and characterization of large cohorts of patients. Extreme caution is required when applying the estimated prognosis to individual patients.

The IMPACT model is also presented as a simple score chart for sequential application of the models. The IMPACT CORE model includes the following variables: age, motor score and pupils. The CORE+ CT model in addition takes into account hypoxia, hypotension, CT classification, tSAH on CT and epidural mass on CT. The CORE+ CT+LAB model in addition takes into account Blood Hb and Blood Glucose. This score chart can be used to obtain an approximate prediction in individual patients. The predictive risk can then be derived by reading the predicted probability from nomograms.

The CRASH trial collaborators and the IMPACT investigators reciprocally validated their prognostic models externally on the other data set and confirmed good performance. They found a small but systematic difference between predictive and observed outcome in the

external validation of the CRASH model, particularly in patients originating from low/middle-income countries.

AIM OF THE STUDY

1. To analyze the causes of traumatic brain injury in patients aged 18 to 40 years.
2. To study age as an important prognostic factor in the prediction of outcome in traumatic brain injury.
3. To study the other prognostic factors in traumatic brain injury in patients aged 18 to 40 years and to compare the same with other age groups.
4. To study the outcome of traumatic brain injury in patients aged 18 to 40 years.

MATERIALS AND METHODS

The incidence of traumatic brain injury is higher in young adults when compared to the rest of the population. Being the productive age group this makes a serious socio economic impact over the family, society and nation.

The aim of the study is to analyse the causes of traumatic brain injuries in this age group and also to study the significance of prognostic factors and their impact over the outcome of traumatic brain injury in that age group.

The study was done after getting approval from Ethical Committee of Govt. Rajaji Hospital, Madurai Medical College, Madurai.

To study this, the patients belonging to the age group of 18 to 40 years admitted with traumatic brain injury in the Head Injury Ward, Government Rajaji Hospital, Madurai for a period of January 2010 to December 2011 were selected and the prognostic factors

predicting the outcome of head injury in this age group were studied by comparing to two sets of control populations – one younger group (3 – 17 years) and another older group (>41 years) – admitted with traumatic brain injury in the same institution at the same period.

In both study population and control groups, patients with polytrauma, alcohol intoxication, drug over dosage and patients with cerebro vascular accidents, spontaneous subarachnoid hemorrhage, patients in postictal state and patients with spinal cord injuries were excluded from the study.

The population under study is named Group ‘A’ and the younger control group (3-17 years) is named Group ‘B’ and the older control group (> 41 years) is named Group ‘C’. All the patients admitted in head injury ward are managed on a standardized treatment protocol based on Trauma Coma Data Bank Study with the available facilities in our hospital.

Once the immediate resuscitative measures to ensure protected airway and to establish adequate circulatory function are over, the

detailed history is sought from the patient or the attender and the patient's demographic factors including age, sex and address were recorded.

After general physical examination and after ruling out chest injuries and associated injuries to abdomen, pelvis, spine and long bones, a detailed neurological assessment is made with attention to the following details.

Consciousness: The most important single parameter of neurological examination is the state of consciousness. Currently the grading of the conscious state as per the Glasgow Coma Scale (GCS), developed by Teasdale and Jennett in the year 1974 at Glasgow, is the universally accepted method. This scale was modified in 1977 and is in common use now.

Glasgow Coma Scale :

Eye Opening : 1. No eye opening 2. Opens eyes to pain

3. Opens eyes to voice 4. Spontaneous eye opening

Motor Response : 1. No movement 2. Extensor response

3. Flexor response 4. Withdraws to pain

5. Localizes to pain 6. Obeys commands

Verbal Response : 1. No sounds 2. Incomprehensible sounds

3. Inappropriate words 4. Confused conversation

5. Well oriented speech

The patient is examined vis-à-vis the above list and scores are given. The total value-score indicates the level of consciousness (15 in a fully conscious patient and three in a deeply comatose areflexic patient). Deterioration or improvement can thus be made out at subsequent examinations done regularly at every 4 hours or earlier when needed. GCS-system avoids arbitrary staging and grading of patients and ambiguous terms are carefully avoided. The simplicity of the chosen terms offers a consistent and accurate assessment when performed by staff nurses and resident doctors. The shortcoming of the Glasgow coma scale is that it is not useful when the patient is on endotracheal tube or

tracheostomy or when he is aphasic. When the eyes were closed by edema, the eye opening response was marked as 'C'; if tracheostomy was done the verbal response was marked as 'T'.

Pupils : The size of the pupils in millimeters and their reaction to light both direct and consensual are recorded. Being a sensitive indicator of developing intracranial haematoma, pupillary examination provides important clues to diagnosis and treatment.

Eye Movements : Dysfunction of eye movements are common after structural lesions and can be tested in conscious cooperative patients. In comatose patients 'Doll's eye movement' can be elicited.

Oculo Cephalic Response : It is tested by moving the patient's head on either side and observing the ocular mobility. Before eliciting this, Cervical Spine fractures should be ruled out. It consists of four defined levels : 1. Suppression of eye movements – normal response in conscious patients. 2. Intact response – Bilateral conjugate righting movements 3. Impaired response – Dysconjugate movements of the eyes 4. Absent response.

In this study, the oculo cephalic response or the doll's eye movement was recorded whether present or absent. Impaired response was taken as brain stem function.

Oculo Vestibular Response: The tympanic membrane is inspected and found it is intact and is not obscured by cerumen. All those patients with obvious bleeding through the ears were omitted. Head is rotated to 30 degree on one side and flexed about 30 degree. Ice-cold water is introduced in 20ml increments with the help of syringe. For declaring this response as 'Absent' at least 100 cc should be used.

The response has four defined levels:

- a) Nystagmus to the same side in the normal conscious patients and in lethargic subjects.
- b) Tonic conjugate deviation to the irrigated side.
- c) Dysconjugate response.
- d) No response.

Oculo vestibular response is recorded as “Present or Absent”. Among the oculo cephalic and oculo vestibular response, Oculo vestibular response is more reliable because of its more powerful stimuli. Hence, the eye movement reflexes cannot be declared as absent unless the ocular vestibular response is done.

Other Cranial Nerve Involvement: Anosmia may occur even with minor head injuries. Fractures of the anterior cranial fossa are more often associated with loss of smell and CSF rhinorrhoea. Optic nerve involvement is often unilateral but rarely bilateral. Fifth cranial nerve may be involved in middle fossa fractures. Fractures involving petrous pyramid may manifest with seventh and eighth cranial nerve palsies. Lower cranial nerve palsies are associated with posterior fossa fractures. In such cases a haematoma below the mastoid may be seen as a skin discolouration and is known as Battle’s sign.

Examination Of The Conscious Patient: Apart from routine neurological assessment, patient’s memory regarding the accident and recent events and the duration of retrograde and post-traumatic amnesia

are recorded along with the time of testing. Post-traumatic amnesia is a good index of the severity of the injury and a good guide to the period of rehabilitation necessary before return to full work.

INVESTIGATIONS

Once the general condition of the patient is stabilized and respiration and blood pressure are steady and the clinical evaluation is completed, biochemical and radiological investigations are carried out depending on the need of the patient. Routine biochemical investigations including Blood Hb%, Blood Urea, Blood Sugar, Serum Creatinine, Serum Electrolytes, Bleeding Time and Clotting Time were done for all patients. Other biochemical investigations are done according to the need of individual patient.

CT brain findings are recorded according to Marshall CT Classification. The structural damages are graded as follows for statistical analysis:

STRUCTURAL DAMAGE	COMPUTED TOMOGRAPHY	GRADE
Diffuse Injury I	No visible pathology	I
Diffuse Injury II	Cisterns present, midline shift of 0.5mm and/or lesion densities present or no mass lesion $> 25\text{cm}^3$	II
Diffuse Injury III (swelling)	Cisterns compressed or absent with a midline shift of 0.5mm or no mass lesion $> 25\text{cm}^3$	III
Diffuse Injury IV (shifting)	Midline shift $> 5\text{mm}$ and no mass lesion $> 25\text{cm}^3$	IV
Evacuated mass lesion	Any lesion surgically evacuated	V
Nonevacuated mass lesion	High or mixed density lesion $> 25\text{cm}^3$ not surgically evacuated	VI

CT scans are helpful in assessing the degree of intracranial injury, in predicting outcome, and, if findings are normal, in avoiding unnecessary hospitalization. They are very sensitive to acute hemorrhage or skull fractures and aid in evaluating (1) intracranial hemorrhage, (2) skull fractures, (3) mass effect and midline shift, (4) obliteration of the basal cisterns and (5) evidence of herniation (subfalcine, tonsillar, or

uncal). CT scans cannot diagnose a concussion (which is a clinical diagnosis) and are poor for diagnosing DAI. If DAI has occurred, CT scans may show small hemorrhages in the corpus callosum and cerebral peduncles. In this case, MRI of the brain should be obtained on the elective basis when the patient is clinically stable because no effective treatment of DAI is currently available. MRI is more sensitive for detecting brainstem injuries, posterior fossa lesions and brain edema. As a general rule, a repeat head CT scan is recommended within 4-8 hours of the initial scan in patients with intracranial hemorrhages and/or coagulopathies. A repeat CT brain scan is recommended sooner in patients who are deteriorating neurologically.

ANALYSIS OF PROGNOSTIC FACTORS AND OUTCOME :

All the prognostic factors having significant impact over outcome were recorded and analysed on univariate analysis basis of individual factors with outcome. Single predictors often have insufficient predictive value to distinguish patients who will do well from those who will do poorly. Moreover, patients can have different characteristics that affect

the prognosis in opposite directions. Hence in this study the prognostic factors were studied on multivariate analysis basis also by analyzing the outcome according to Glasgow Coma Scale and Madras Head Injury Prognostic Scale.

Madras Head Injury Prognostic Scale: Ramesh et al developed the Madras Head Injury Prognostic Scale (MHIPS). This can help determine the outcome for a trauma patient with a head injury. This multivariate analysis scale has the following parameters:

1. age of the patient in years
2. best motor response from the Glasgow coma scale
3. pupillary light response
4. oculocephalic response
5. CT scan findings
6. systemic injuries

All the six parameters were carefully analyzed in all the patients and given points as follows:

Parameter	Findings	Points
age of the patient	<15 years	3
	15 to 45 years	2
	>45 years	1
best motor response	5 or 6	3
	3 or 4	2
	1 or 2	1
pupillary light response	normal	3
	impaired	2
	absent	1
oculocephalic response	normal	3
	impaired	2
	Absent	1

CT scan findings	normal	3
	partially effaced basal cisterns OR midline shift <5 mm OR lesion density <3 cm	2
	absent basal cisterns OR midline shift ≥ 5 mm OR lesion density ≥ 3 cm	1
systemic injuries	no other injuries	3
	1 or 2 long bone fractures	2
	3 or more long bone fractures OR visceral injuries (thoracic, abdominal, pelvic)	1

Total score = SUM (points for all 6 parameters)

For analyzing the outcome, the patients are grouped under one of the three groups according to the sum score of MHIPS as follows:

1. MHIPS 15-18
2. MHIPS 13 -14
3. MHIPS 6-12

MHIPS sum score of 15-18 is predicted with good outcome, score of 13-14 is predicted with poor outcome and score of 6-12 is predicted with death.

RESULTS OF THE STUDY

TABLE: 1. AGE

Age in years	Group A	Group B	Group C	Total
Range	18 to 40	3 to 17	41 to 84	3 to 84
Mean	29.6	12.3	62.6	35.8
S.D.	6.4	3.7	10.4	19.5
‘p’	0.0001 Significant			

In the study Group, the mean age is 29.6 with SD of 6.4; in Group B mean age is 12.3 and SD is 3.7 and in Group C mean age is 62.6 and SD is 10.4 (p= 0.0001 significant).

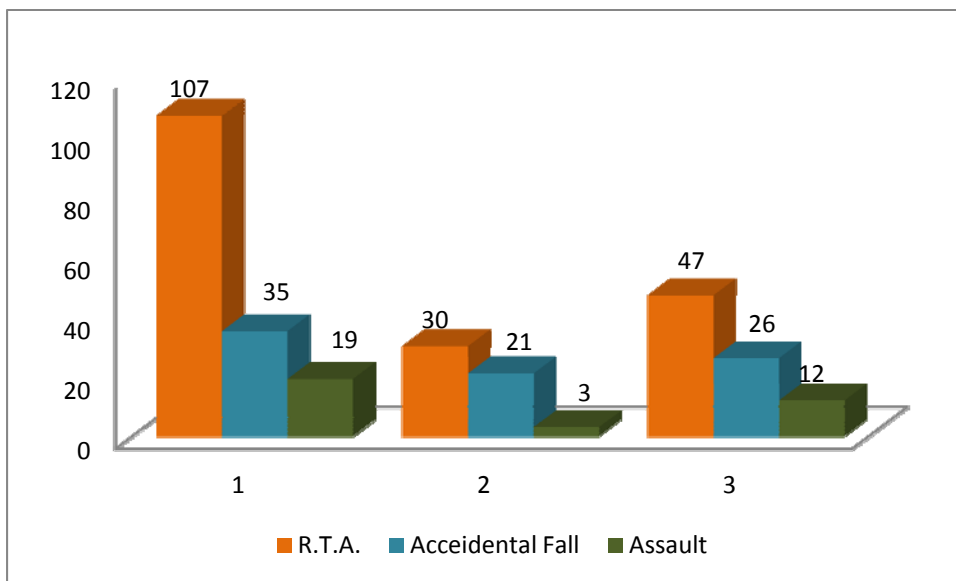
TABLE: 2. SEX

SEX	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Male	128	79.5	38	70.4	60	70.6	217	72.3
Female	33	20.5	16	29.6	25	29.4	83	27.7
TOTAL	161	100	54	100	85	100	300	100

The ratio of males to females in the study Group is 3.88:1, while it is 2.38:1 in Group B and 2.4:1 in Group C. Among the 161 patients in Group A 128 were males and 33 were females. In Group B there were 38 males and 16 females. Of the 85 patients in Group C, 60 were males and 25 females.

TABLE: 3. CAUSES OF TBI

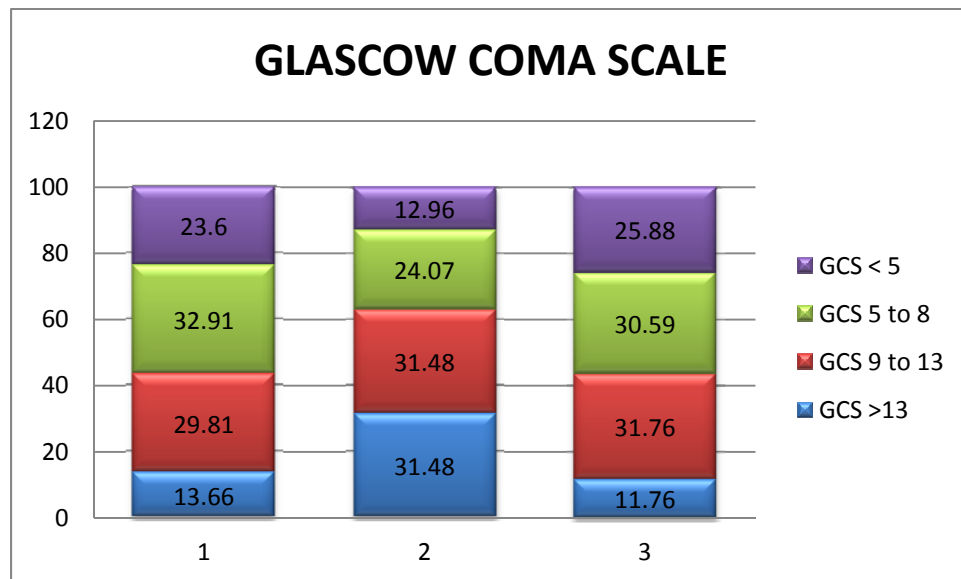
CAUSES OF TBI	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
R.T.A.	107	66.46	30	55.56	47	55.29	184	61.33
Accidental Fall	35	21.74	21	38.89	26	30.59	82	27.33
Assault	19	11.8	3	5.55	12	14.12	34	11.33
Total	161	100	54	100	85	100	300	100



In Group A, the commonest cause of TBI is RTA(66.46%) followed by falls(21.74%) and assaults(11.8%). TBI due to fall is more in Group C (30.59%) than the study Group (21.74%). In Group C, RTA constitutes 55.56% of TBI while falls account for 38.89%.

TABLE: 4. GLASCOW COMA SCALE

Glasgow Coma Scale	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
>13	22	13.66	17	31.48	10	11.76	49	16.33
9 to 13	48	29.81	17	31.48	27	31.76	92	30.67
5 to 8	53	32.91	13	24.07	26	30.59	92	30.67
< 5	38	23.6	7	12.96	22	25.88	67	22.33
TOTAL	161	100	54	100	85	100	300	100



Group A Group B Group C

Values in percentage

TABLE: 5. PUPILLARY REACTION

Pupillary Reaction	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Normal	81	50.31	43	79.63	36	42.35	160	53.33
Abnormal	80	49.69	11	20.37	49	57.65	140	46.67
TOTAL	161	100	54	100	85	100	300	100

The pupillary reaction is normal in 50.31% cases in Group A, 79.63% cases in Group B and 42.35% cases in Group C. The abnormal pupillary reaction is observed in 49.69% cases of Group A, 20.37% cases of Group B and 57.65% cases of Group C.

TABLE: 6. OCULOCEPHALIC REFLEX

OCR	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Present	126	78.26	48	88.89	57	67.06	231	77
Absent	35	21.74	6	11.11	28	32.94	69	23
TOTAL	161	100	54	100	85	100	300	100

The oculocephalic response is present in 78.26% of cases in Group A and absent in 21.74% of cases. In Group B, OCR is present in 88.89% of cases and absent in 11.11% of cases. In Group C, the same is present in 67.06% of cases and absent in 32.94% of cases.

TABLE: 7. OCULOVESTIBULAR REFLEX

OVR	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Yes	130	80.75	50	92.6	59	69.41	239	79.67
No	31	19.25	4	7.4	26	30.59	61	20.33
TOTAL	161	100	54	100	85	100	300	100

OVR is present in 80.75% in Group A; 92.6% in Group B and 69.41% in Group C. The same is absent in 19.25% in Group A; 7.4% in Group B and 30.59% in Group C.

TABLE: 8. C.T. BRAIN FINDINGS

MARSHALL C.T. Findings	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Diffuse Injury I	42	26.1	16	29.6	23	27.1	81	27
Diffuse Injury II	24	14.9	8	14.8	10	11.7	42	14
Diffuse Injury III	22	13.7	6	11.1	8	9.4	36	12
Diffuse Injury IV	42	26.1	10	18.5	18	21.2	70	23.3
Evacuated mass lesion	22	13.7	11	20.4	18	21.2	51	17
Non Evacuated Mass Lesion	9	5.6	3	5.6	8	9.4	20	6.7
Total	161	100	54	100	85	100	300	100

Diffuse Injury I is seen in 26.1% of Group A , 29.6% of Group B, 27.1% of Group C. Diffuse Injury II is seen in 14.9 % of Group A , 14.8% of Group B, 11.7% of Group C. In the study evacuated mass lesion constitute 13.7% cases in Group A, 20.4% cases in Group B and 21.2% cases in Group C. Diffuse injury IV is seen in 26.1% of Group A cases, 18.5% cases in Group B and 21.2% in Group C.

TABLE: 9. MADRAS HEAD INJURY PROGNOSTIC SCALE

MHIPS	GROUP A		GROUP B		GROUP C	
	No	%	No`	%	No	%
15 To 18	58	36	36	66.7	15	17.6
13 To 14	38	23.6	10	18.5	35	41.2
6 To 12	65	40.4	8	14.8	35	41.2
TOTAL	161	100	54	100	85	100

In Group A, 36% cases belong to MHIPS 15 to 18, while the case is 66.7% in Group B and 17.6% in Group C. In Group A, 23.6% cases belong to MHIPS 13 to 14, while the case is 18.5% in Group B and 41.2% in Group C. In Group A, 40.4% cases belong to MHIPS 6 to 12, while the case is 14.8% in Group B and 41.2% in Group C.

TABLE: 10. TREATMENT

Treatment	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Conservative management	90	55.9	29	53.7	50	58.82	169	56.33
Surgical management	71	44.1	25	46.3	35	41.17	131	43.66
Total	161	100	54	100	85	100	300	100

In the study 90 patients in Group A were treated conservatively and 71 underwent surgery; 29 patients in Group B were treated conservatively and 25 underwent surgery; 50 patients in Group C treated conservatively and 16 patients underwent surgery.

DISCUSSION

In the study, 49.06% of Group A patients, 68.52% of Group B and 38.82% of Group C patients had good outcome. The outcome is poor in 13.66% of Group A, 14.81% of Group B and 12.94% of Group C. The mortality is 37.26% in Group A, 16.67% in Group B and 48.24% in Group C.

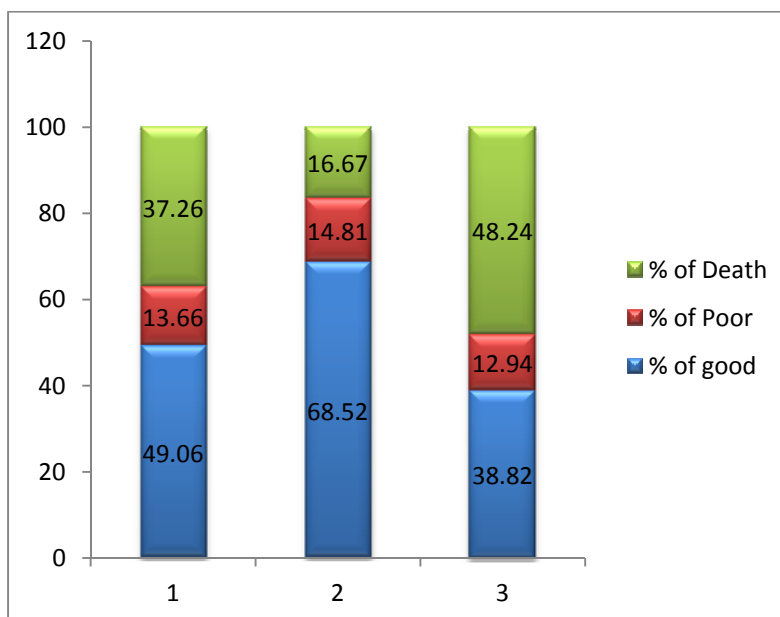
ANALYSIS OF CAUSES OF TBI

In the population under study, RTA is the commonest cause TBI (66.46%) followed by falls (21.74%) and assaults (19%) which correlates with many previous studies.

In RTA, majority of the victims were motor-cyclists and pedestrians. The patients in Group C often had lower impact injuries. The percentage of falls (30.59%) is more in these groups than the study group (21.74%) which also correlates with previous studies.

TABLE: 11. GLASCOW OUTCOME SCALE

Glasgow Outcome Scale	Group A		Group B		Group C		Total	
	No.	%	No.	%	No.	%	No.	%
Good	79	49.06	37	68.52	33	38.82	149	49.67
Poor	22	13.66	8	14.81	11	12.94	41	13.67
Death	60	37.26	9	16.67	41	48.24	110	36.66
TOTAL	161	100	54	100	85	100	300	100



Group A

Group B

Group C

TABLE: 12. CAUSES OF TBI AND OUTCOME

CAUSE OF TBI	Group A				Group B				Group C				Total			
	Good		Poor & Death		Good		Poor & Death		Good		Poor & Death		Good		Poor & Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
RTA	53	49.5	54	50.5	20	66.7	10	33.3	16	34.0	31	66.0	89	48.4	95	51.6
Fall	15	42.8	20	57.1	14	66.7	7	33.3	11	42.3	15	57.7	40	48.8	42	51.2
Assault	11	57.8	8	42.1	3	100	0	0	6	50	6	50	20	58.8	14	41.2

ANALYSIS OF PROGNOSTIC FACTORS AND OUTCOME

DEMOGRAPHIC FACTORS:

1. AGE & OUTCOME:

The favorable outcome in TBI in study population (49.1%) and younger control population (68.5%) is significantly greater than older control population (38.8%) which correlates with previous studies. Most of the studies confirm that age is a significant prognostic factor in deciding the outcome in TBI. Mortality is also very high in the elderly age group in many series¹⁴. The study shows a statistically significant relationship between age and outcome ($p=0.0006$ significant). When the GCS on admission was taken into account there is a trend of better outcome at all levels in study population compared to older population. Moreover due to co-morbid conditions and their complications, the Group C population had a poor outcome and high rate of mortality than the younger cohorts. In this study, 61.2% of Group C patients had unfavorable outcome when compared to Group A (50.9%) and Group B (31.5%). The same picture is reflected in the works of Caresson et al and Becker et al¹⁰

2.SEX AND OUTCOME:

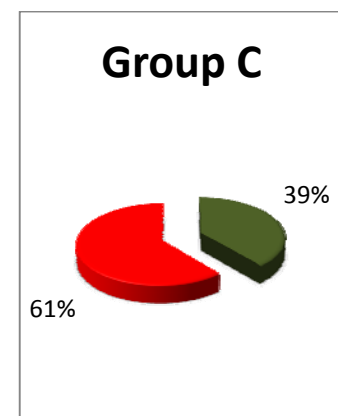
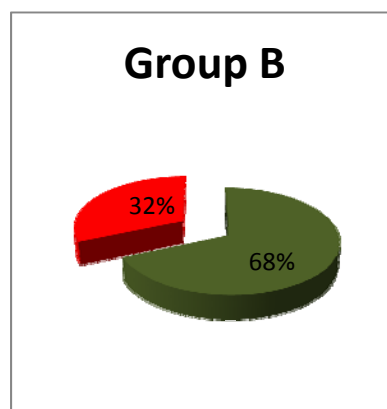
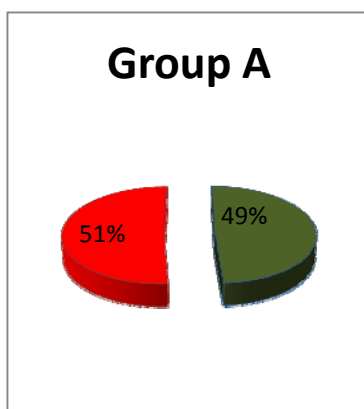
In Group A, among males 47.7% had good outcome and 52.3% had poor outcome whereas among females 54.5% had good outcome and 45.5% had poor outcome. In Group B, among males 76.3% had good outcome and 23.7% had poor outcome whereas among females 50% had good outcome and 50% had poor outcome. In Group C, among males 42% had good outcome and 58.3% had poor outcome whereas among females 32% had good outcome and 68% had poor outcome. There is no significant statistical correlation between sex and outcome in this study.

TABLE: 13. AGE AND OUTCOME

Outcome	Group A		Group B		Group C		Total	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Good	29.2	6.6	12.1	4	60.8	11.5	32	18.5
Bad (Poor & Death)	29.9	6.3	12.7	3.3	63.8	9.7	39.7	19.8
P'	0.0006 Significant							

TABLE: 14. AGE AND OUTCOME

OUTCOME	GROUP A		GROUP B		GROUP C		TOTAL	
	No	%	No	%	No	%	No	%
GOOD	79	49.1	37	68.5	33	38.8	149	49.7
BAD (Poor & Death)	82	50.9	17	31.5	52	61.2	151	50.3
TOTAL	161	100	54	100	85	100	300	100



Good
 Poor & death

TABLE: 15. SEX AND OUTCOME

SEX	GROUP A				GROUP B				GROUP C				TOTAL			
	GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH	
	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%
MALE	61	47.7	67	52.3	29	76.3	9	23.7	25	42	35	58.3	115	50.9	111	49.1
FEMALE	18	54.5	15	45.5	8	50	8	50	8	32	17	68	34	45.9	40	54.1

CLINICAL SEVERITY AND OUTCOME

1. GCS & OUTCOME:

Although GCS is not intended to use as a prognostic indicator, the depth and duration of coma is related to the outcome. In general, there was a strong correlation between decreasing GCS and increasing mortality, whether the observation was made in the emergency room after resuscitation or after 24 hrs from the time of admission. In Group A 72.9% of mild and moderately severe TBI had good outcome whereas it is 91.2% in Group B and 56.8% in Group C. In severe and critical TBI according to GCS, 69.2% of Group A, 70% of Group B and 75% of Group C had unfavorable outcome. There is a strong correlation between age and outcome in all grades of clinical severity of TBI according to GCS, old age had a relatively poor outcome in all grades of GCS than their younger cohorts.

Analyzing the outcome, there is strong statistical significance in all age groups with increasing clinical severity of TBI as assessed by GCS and outcome. [$p=0.0001$ (significant) in Group A; $p=0.0001$ (significant)

in Group B and $p=0.0059$ (significant) in Group C]. These results also correlate with previous studies.

2. PUPILLARY REACTION AND OUTCOME

There was strong correlation between bilateral absence of pupillary light response and poor outcome following severe traumatic brain injury in all age groups. In Group A 61.7% of normal pupillary response had good outcome. The case is so in 76.7% of Group B patients and 55.6% of Group C patients. In Group A 63.7% of abnormal pupillary response had poor outcome. The case is so in 63.6% of Group B patients and 73.5% of Group C patients. There is strong statistical correlation between the pupillary reaction and outcome in all age groups ($p=0.0021$ significant in Group A, $p=0.0156$ significant in Group B and $p=0.0128$ significant in Group C).

TABLE: 16. GCS AND OUTCOME

GCS	GROUP A				GROUP B				GROUP C				TOTAL			
	GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
9 to 15 (Mild & Moderate)	51	72.9	19	27.1	31	91.2	3	8.8	21	56.8	16	43.2	103	73.1	38	26.9
3 to 8(severe& critical)	28	30.8	63	69.2	6	30	14	70	12	25	36	75	46	28.9	113	80.1
p	0.0001 Significant				0.0001 Significant				0.0059 Significant				0.0001 Significant			

GCS AND OUTCOME

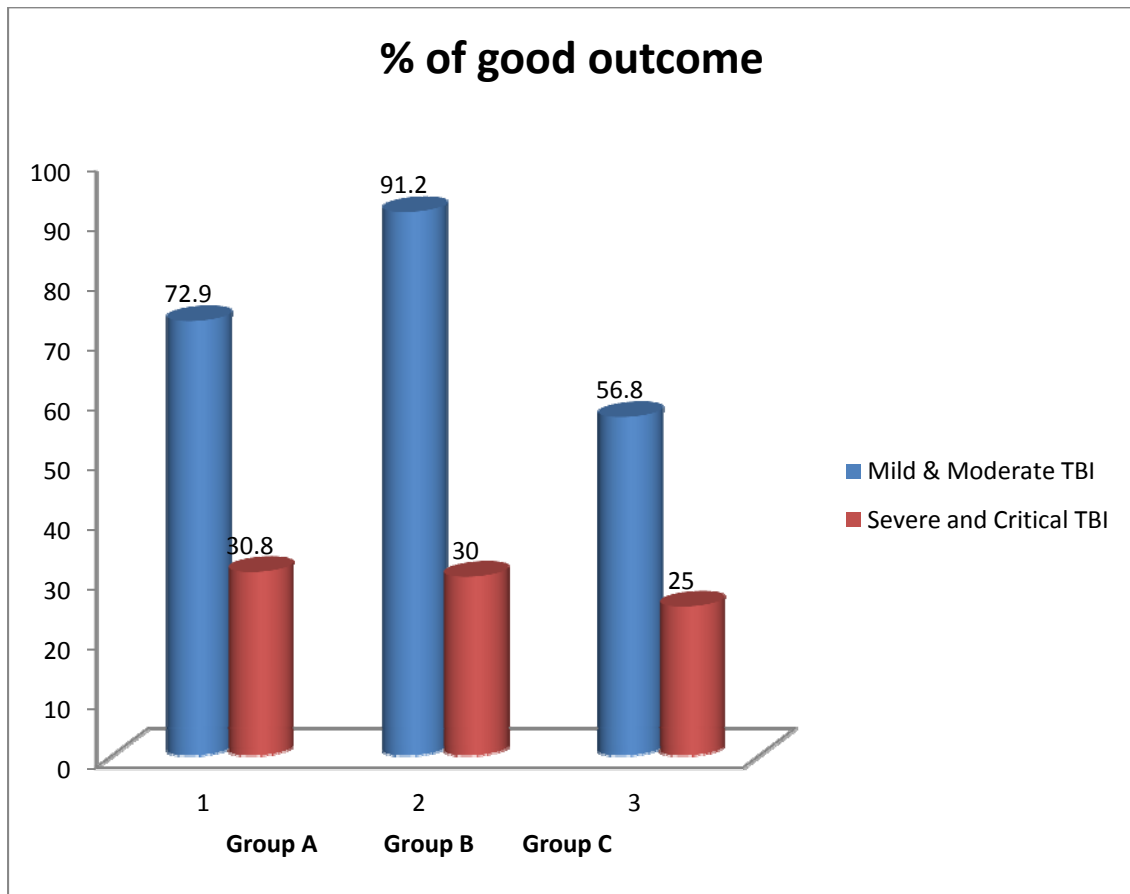
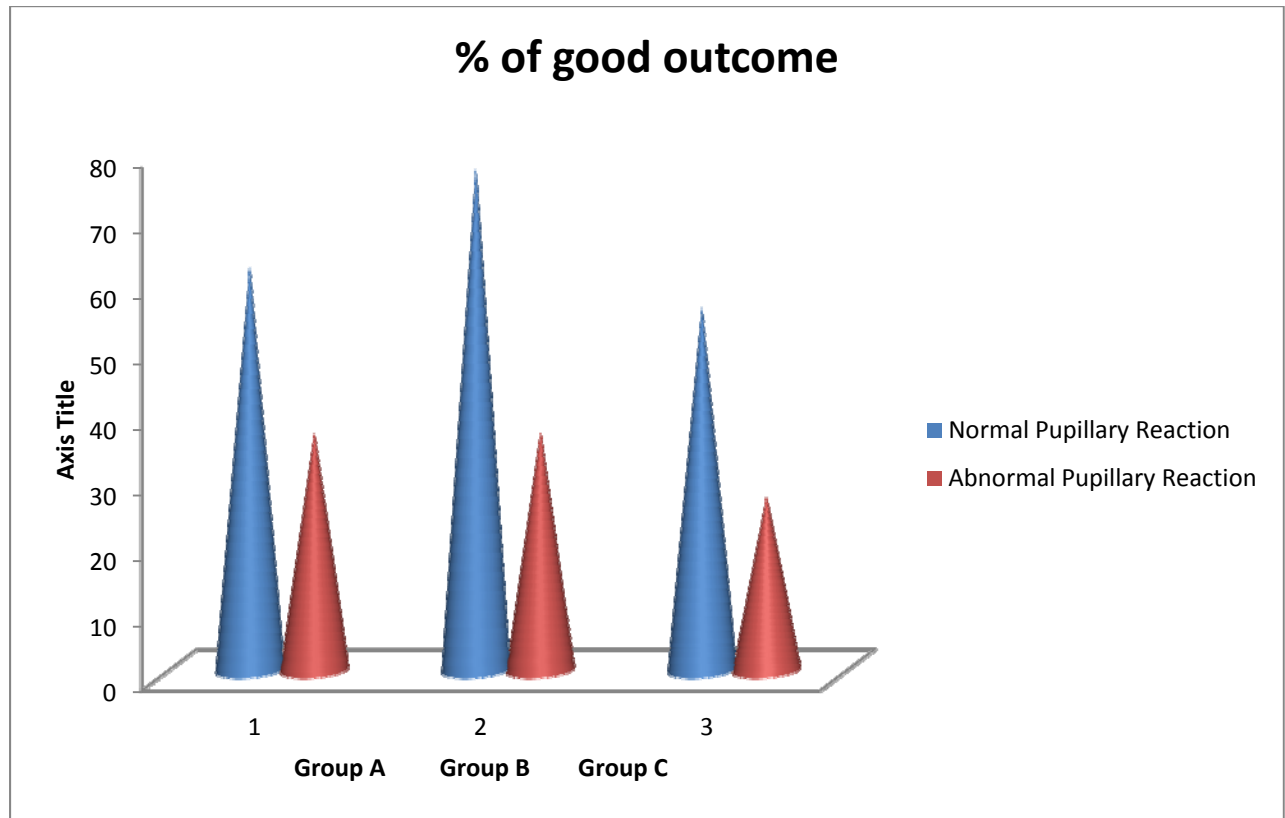


TABLE: 17. PUPLLIARY REACTION AND OUTCOME

Puplliary Reaction	Group A				Group B				Group C				Total			
	Good		Poor & Death		Good		Poor & Death		Good		Poor & Death		Good		Poor & Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Normal	50	61.7	31	38.2	33	76.7	10	23.3	20	55.6	16	44.4	103	64.4	57	35.6
Abnormal	29	36.3	51	63.7	4	36.4	7	63.6	13	26.5	36	73.5	46	32.9	94	67.1
P'	0.0021 Significant				0.0156 Significant				0.0128 Significant				0.0001 Significant			

. PUPILLIARY REACTION AND OUTCOME



3. OCR AND OUTCOME:

The study clearly shows that OCR is one of the powerful predictors of outcome. The presence of OCR favors a good outcome in all age groups (59.5% in Group A, 75% in Group B and 50.9% in Group C). In Group A 88.6% without OCR had poor outcome ($p=0.0001$ significant). A similar statistical significance is seen in Group B ($p=0.0093$ significant) and Group C ($p=0.0026$ significant).

4. OVR AND OUTCOME:

Likewise, the presence of OVR favours a good outcome in all age groups (59.2% in Group A, 72% in Group B and 50.8% in Group C). 93.5% of cases in Group A without OVR had poor outcome ($p=0.0001$ significant). A similar statistical significance is seen in Group B ($p=0.0071$ significant) and Group C ($p=0.0006$ significant).

TABLE: 18. OCR AND OUTCOME

OCR	Group A				Group B				Group C				Total			
	Good		Poor & Death		Good		Poor & Death		Good		Poor & Death		Good		Poor & Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	75	59.5	51	40.5	36	75	12	25	29	50.9	28	49.1	140	60.6	91	39.4
No	4	11.4	31	88.6	1	16.7	5	83.3	4	14.3	24	85.7	9	13	60	87
P'	0.0001 Significant				0.0093 Significant				0.0026 Significant				0.0001 Significant			

TABLE: 19. OVR AND OUTCOME

OVR	Group A				Group B				Group C				Total			
	Good		Poor & Death		Good		Poor & Death		Good		Poor & Death		Good		Poor & Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	77	59.2	53	40.8	36	72	14	28	30	50.8	29	49.2	143	59.8	96	40.2
No	2	6.5	29	93.5	1	25	3	75	3	11.5	23	88.5	6	9.8	55	90.2
P'	0.0001 Significant				0.0071 Significant				0.0006 Significant				0.0001 Significant			

ANALYSIS OF STRUCTURAL ABNORMALITIES

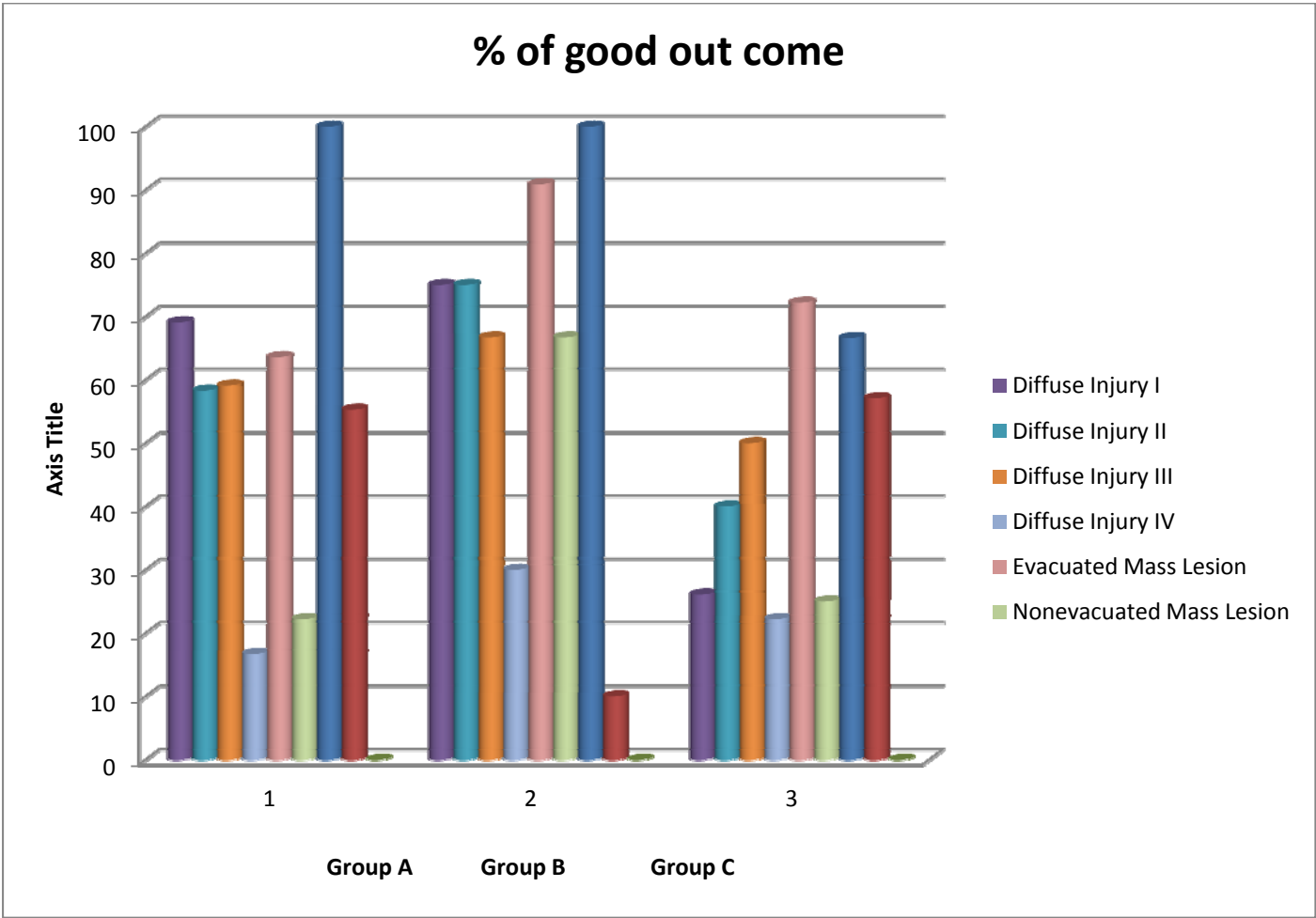
1. MARSHALL CT CLASSIFICATION AND OUTCOME:

Patients with Diffuse Injury IV had poor outcome in all age Groups, which correlates with previous studies. 83.3% of Group A, 70% of Group B and 77.8% of Group C belonging to Diffuse Injury IV ended with poor outcome. The non evacuated mass lesions with midline shift also had a poor outcome (77.8% in Group A, 33.3% in Group B and 75% in Group C). Those with Diffuse Injury I and II had a favorable outcome. In diffuse injury I, 69.1% of Group A and 75% Of Group B had a good outcome whereas only 26.1% of Group C had a favorable outcome because of the increased prevalence of co-morbid illness in elderly.

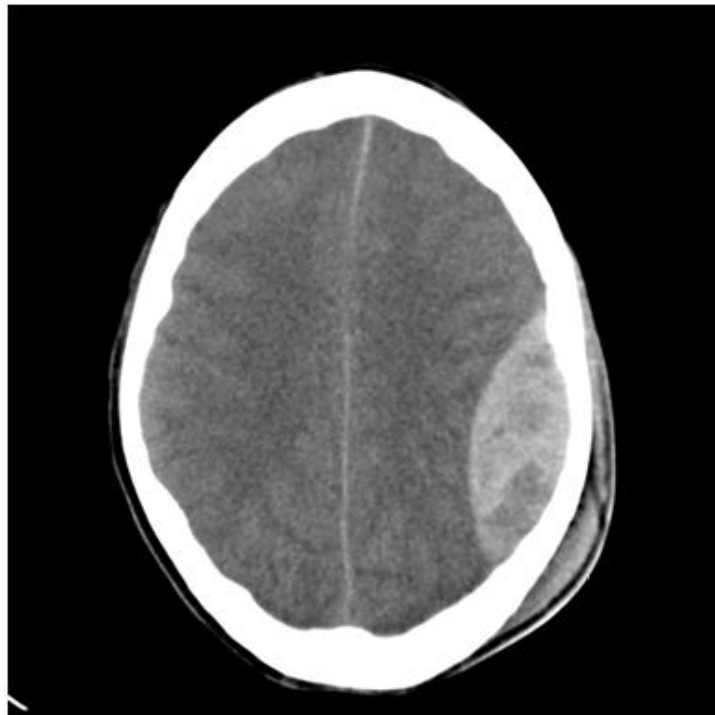
TABLE: 20. MARSHALL CT CLASSIFICATION AND OUTCOME

MARSHALL CT CLASSIFICATION	Group A				Group B				Group C				Total			
	GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH		GOOD		POOR& DEATH	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
Diffuse injury I	29	69.1	13	30.9	12	75	4	25	6	26.1	17	73.9	47	58	34	42
Diffuse injury ii	14	58.3	10	41.7	6	75	2	25	4	40	6	60	24	57.1	18	42.9
Diffuse injury iii	13	59.1	9	40.9	4	66.7	2	33.3	4	50	4	50	21	58.3	15	41.7
Diffuse injury iv	7	16.7	35	83.3	3	30	7	70	4	22.2	14	77.8	14	20	56	80
Evacuated mass lesion	14	63.6	8	36.4	10	90.9	1	9.1	13	72.2	5	27.8	37	72.5	14	27.5
Non evacuated mass lesion	2	22.2	7	77.8	2	66.7	1	33.3	2	25	6	75	6	30	14	70

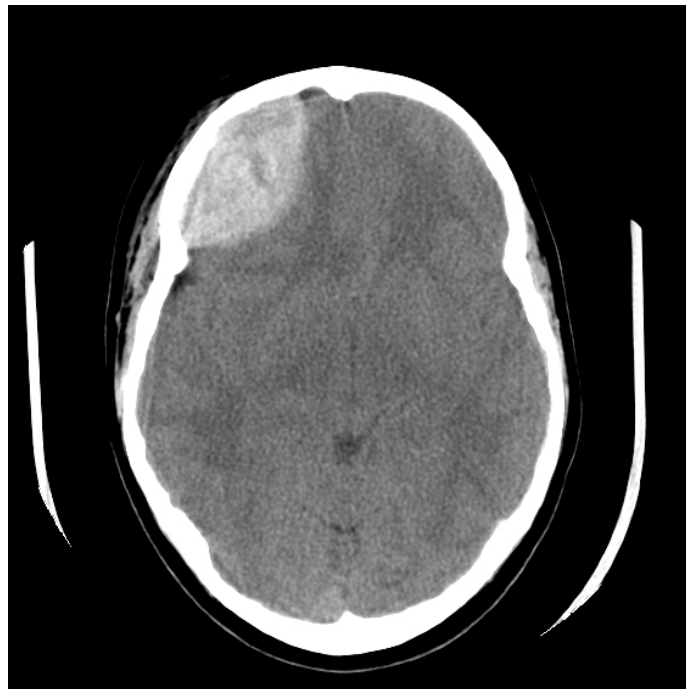
MARSHALL CT CLASSIFICATION AND OUTCOME



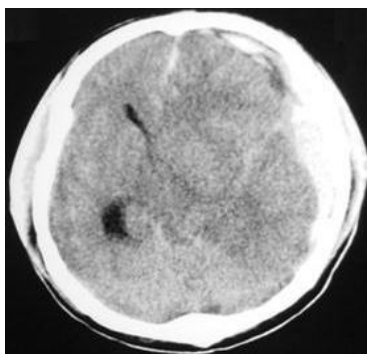
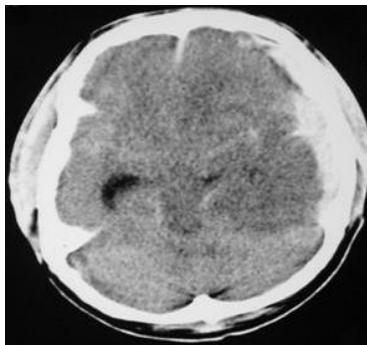
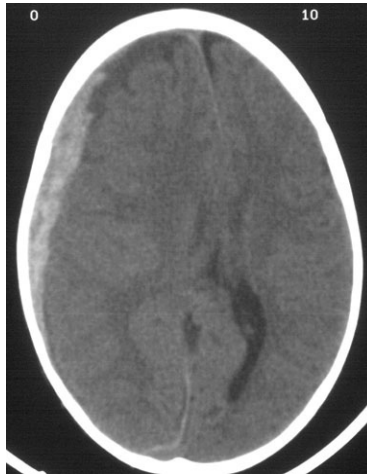
INTRACRANIAL MASS LESIONS EVACUATED



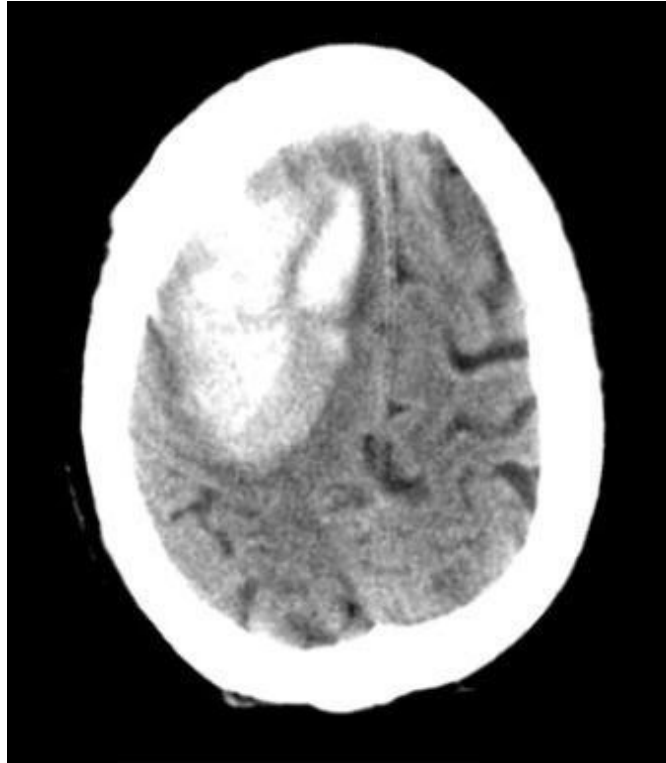
LEFT PARIETAL EXTRA DURAL HAEMATOMA



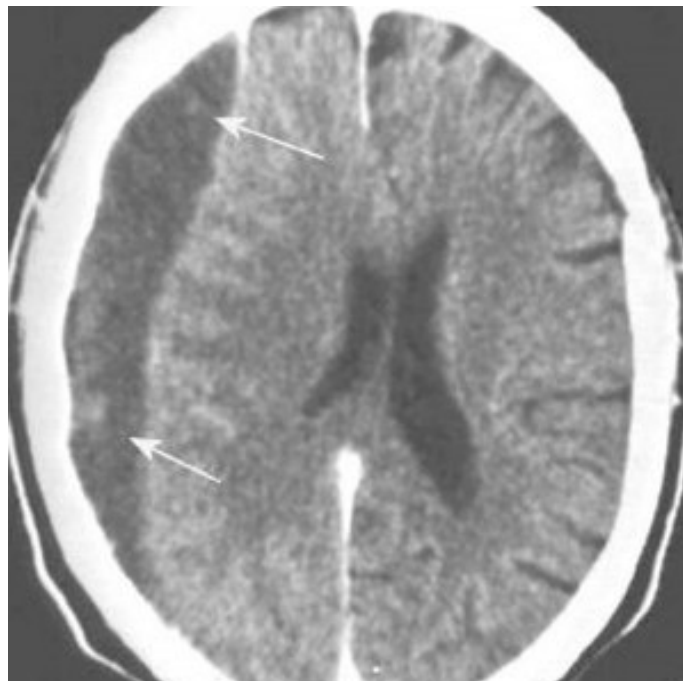
RIGHT FRONTAL EXTRA DURAL HAEMATOMA
INTRACRANIAL MASS LESIONS EVACUATED
ACUTE SUBDURAL HAEMATOMA



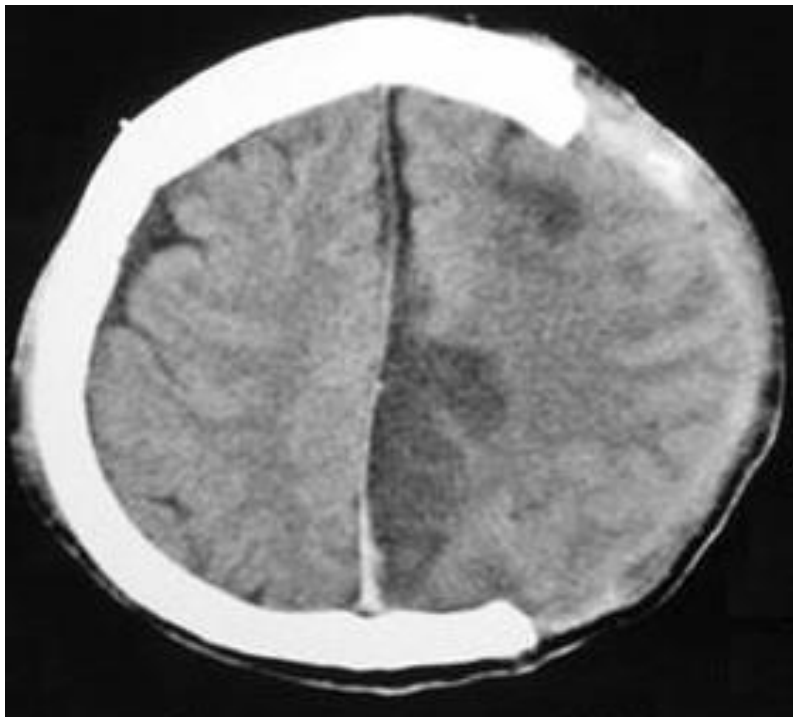
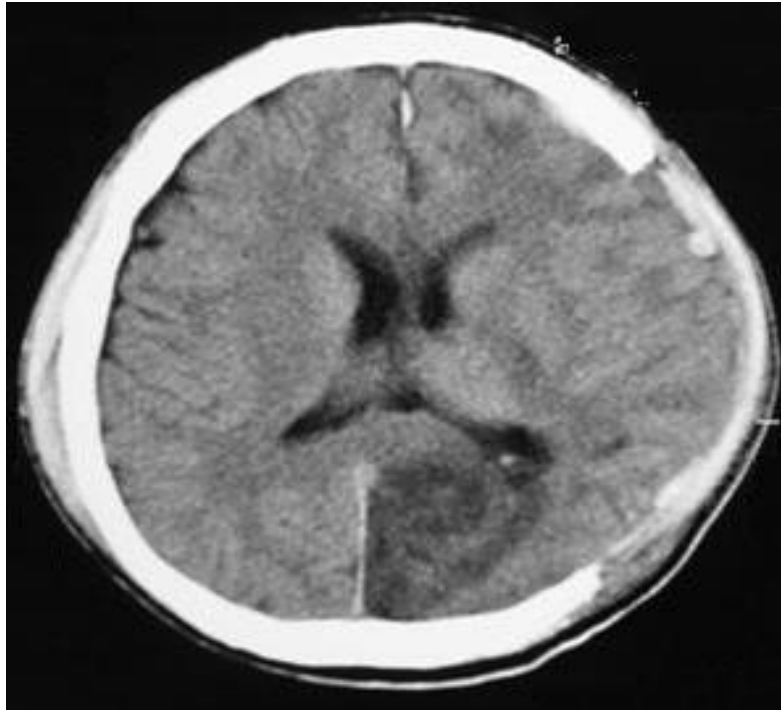
INTRACRANIAL MASS LESIONS EVACUATED



INTRACEREBRAL HAEMATOMA



CHRONIC SUBDURAL HAEMATOMA
POST OPERATIVE CT SCAN
AFTER DECOMPRESSIVE CRANIECTOMY



MULTIVARIATE ANALYSIS

MHIPS AND OUTCOME:

Patients with MHIPS 15-18 had good outcome in all age groups (100% in Group A, 100% in Group B and 86.6% in Group C).

In patients admitted with MHIPS 13- 14, in Group A 36.8% had poor outcome and 7.9% had mortality. In Group B 80% had poor outcome and 10% had mortality. In Group C 20% had poor outcome and 22.9% had mortality.

Patients with MHIPS 6-12 had increased mortality in all age groups (87.7% in Group A, 100% in Group B and 91.4% in Group C).

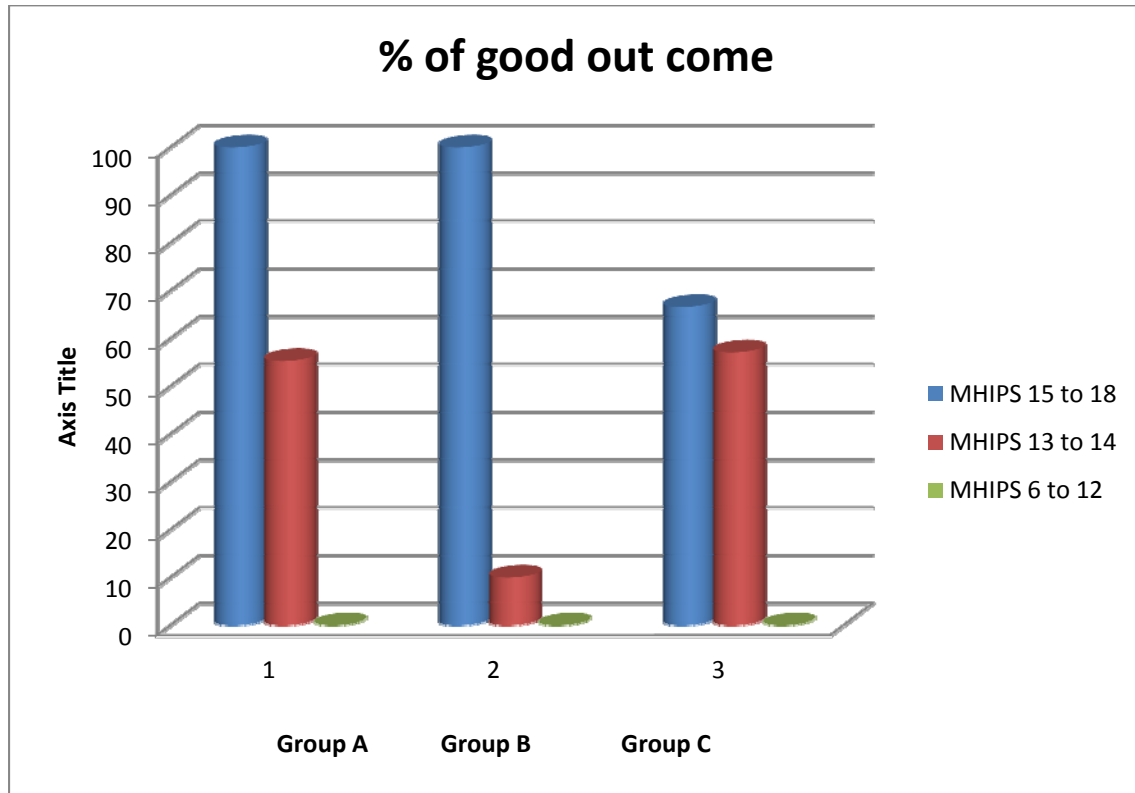
These results correlate well with previous studies in predicting outcome especially in MHIPS 15 to 18 and MHIPS 6 to 12. These results also clearly show the significance of the following clinical prognostic factors in predicting the outcome: age, motor response, pupillary reaction and oculocephalic reflex.

TABLE : 21. MHIPS AND OUTCOME

MHIPS	GROUP A						GROUP B						GROUP C						TOTAL					
	Good		Poor		Death		Good		Poor		Death		Good		Poor		Death		Good		Poor		Death	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
15 to 18	58	100	0	0	0	0	36	100	0	0	0	0	13	86.6	1	6.7	1	6.7	107	98.2	1	0.9	1	0
13 to 14	21	55.3	14	36.8	3	7.9	1	10	8	80	1	10	20	57.1	7	20	8	22.9	42	50.6	29	34.9	12	14.3
6 to 12	0	0	8	12.3	57	87.7	0	0	0	0	8	100	0	0	3	8.6	32	91.4	0	0	11	10.2	97	89.8

MHIPS AND

OUTCOME



TREATMENT AND OUTCOME:

In Group A, 90 patients were treated conservatively and 71 patients underwent surgery. In Group B, 29 patients were treated conservatively and 25 patients underwent surgery. In Group C, 50 patients were treated conservatively and 35 patients underwent surgery.

In patients managed conservatively there is a significant correlation between age and outcome. Favorable outcome is noticed in 45.6% of cases in Group A and 55.2% of cases in Group B, whereas only 24% of Group C patients managed conservatively had a good outcome, mainly due to existence of co morbid illnesses in elderly patients.

In patients who underwent surgery, a favorable outcome is noticed in 53.5% of Group A patients, 84% of Group B patients and 60% of Group C patients. In Group A 46.5% of cases operated had a poor outcome mainly due to late referrals from primary level referral centres.

Combining both conservative and surgical management, good outcome is seen in 49.06% of Group A, 68.52% of Group B and 38.82% of Group C patients reflecting strongly that age is a significant prognostic factor in predicting the outcome in traumatic brain injury.

TABLE: 22. TREATMENT AND OUTCOME

Treatment	Group A				Group B				Group C				Total			
	Good		Poor & Death		Good		Poor & Death		Good		Poor & Death		Good		Poor & Death	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Conservative management	41	45.6	49	54.4	16	55.2	13	44.8	12	24	38	76	69	40.8	100	59.2
Surgical management	38	53.5	33	46.5	21	84	4	16	21	60	14	40	80	61.1	51	38.9

CONCLUSION

The study clearly showed that age is one of the strongest predictors of outcome after TBI, with the study population having a better outcome in all grades of severity of TBI than the older control population. Among the study population the commonest cause of TBI is RTA followed by falls and assaults.

Among the various prognostic factors studied, age, motor score and pupillary reactivity bear a strong relationship with outcome as evidenced by univariate and multivariate analysis. The mild and moderate TBI according to Glasgow Coma Scale have a better prognosis than severe and critical TBI in all age groups. In the study group, patients with MHIPS of 15 - 18 had favorable outcome while those with <13 had unfavorable outcome. Bilateral absence of pupillary light reflex and impaired or absent oculocephalic response & oculovestibular response predicted a poor outcome. Diffuse injury IV of Marshall CT classification significantly correlated with poor outcome.

This study confirms that the largest amount of prognostic information regarding outcome in the study group is contained in the core set 3 predictors namely age, motor score and pupillary reactivity. Better understanding of these factors will help to improve the quality of care provided to patients with traumatic brain injury.

BIBLIOGRAPHY

1. Alexander S, Kerr ME, Kim Y, et al: Apolipoprotein E4 allele presence and functional outcome after severe traumatic brain injury. *J Neurotrauma* 2007; 24:790-797.
2. Association for the Advancement of Automotive Medicine : *The Abbreviated Injury Scale, 1990 revision*. Des Plaines, IL, Association for the Advancement of Automotive Medicine, 1990.
3. Baker SP, O'Neill B, Haddon Jr W, et al: The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. *J Trauma* 1974; 14:187-196.
4. Balestreri M, Czosnyka M, Chatfield DA, et al: Predictive value of Glasgow Coma Scale after brain trauma: change in trend over the past ten years. *J Neurol Neurosurg Psychiatry* 2004; 75:161-162.
5. Berger MS, Pitts LH, Lovely M, et al: The outcome from severe head injury in children and adolescents. *J Neurosurg* 1985; 62:194-199.

6. Buechler CM, Blostein PA, Koestner A, et al: Variation among trauma centers' calculation of Glasgow Coma Scale score: results of a national survey. *J Trauma* 1998; 45:429-432.
7. Burnett DM, Kolakowsky-Hayner SA, Slater D, et al: Ethnographic analysis of traumatic brain injury patients in the national Model Systems database. *Arch Phys Med Rehabil* 2003; 84:263-267.
8. Butcher I, McHugh GS, Lu J, et al: Prognostic value of cause of injury in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:281-286.
9. Butcher I, Maas AI, Lu J, et al: Prognostic value of admission blood pressure in traumatic brain injury; results from the IMPACT study. *J Neurotrauma* 2007; 24:294-302.
10. Carlsson CA, von Essen I, Fugner J: factors affecting the clinical course the patients with severe head trauma: *J. Neu. Surg.*, 29:242-252, 1968.

11. Combes P, Fauvage B, Colonna M, et al: Severe head injuries: an outcome prediction and survival analysis. *Intensive Care Med* 1996; 22:1391-1395.
12. Farace E, Alves WM: Do women fare worse: a metaanalysis of gender differences in traumatic brain injury outcome. *J Neurosurg* 2000; 93:539-545.
13. Flint AC, Manley GT, Gean AD, et al: Post-operative expansion of hemorrhagic contusions after unilateral decompressive craniectomy in severe traumatic brain injury. *J Neurotrauma* 2008; 25:503-512.
14. Gudeman S K , Kishore K PR , Miller J D ,et al : .Neu. Sur. 5 : 309-313, 1979
15. Hukkelhoven CW, Steyerberg EW, Rampen AJ, et al: Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients. *J Neurosurg* 2003; 99:666-673.
16. Jennett V , Teasdale G , Braakman R, Minderhoud J, Krill Jones R: predicting outcome in individual patients after severe head injury. *Lancet*, 1 : 1031-1034, 1976.

17. Jordan BD: Genetic influences on outcome following traumatic brain injury. *Neurochem Res* 2007; 32:905-915.
18. Jorge RE, Robinson RG, Starkstein SE, et al: Influence of major depression on 1-year outcome in patients with traumatic brain injury. *J Neurosurg* 1994; 81:726-733.
19. Kakarieka A, Braakman R, Schakel EH: Clinical significance of the finding of subarachnoid blood on CR scan after head injury. *Acta Neurochir (Wien)* 1994; 129:1-5.
20. Lavati A, Farina ML, Vecchi G, et al: Prognosis of severe head injuries. *J Neurosurg* 1982; 57:779-783.
21. Maas AI, Steyerberg EW, Butcher I, et al: Prognostic value of computerized tomography scan characteristics in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:303-314.
22. Maas AI, Hukkelhoven CW, Marshall LF, et al: Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic

classification and combinations of computed tomographic predictors. *Neurosurgery* 2005; 56:1173-1182.

23. Manley G, Knudson MM, Morabito D, et al: Hypotension, hypoxia, and head injury: frequency, duration, and consequences. *Arch Surg* 2001; 136:1118-1123.
24. Marmarou A, Lu J, Butcher I, et al: IMPACT database of traumatic brain injury: design and description. *J Neurotrauma* 2007; 24:239-250.
25. Marmarou A, Lu J, Butcher I, et al: Prognostic value of the Glasgow Coma Scale and pupil reactivity in traumatic brain injury assessed pre-hospital and on enrollment: an IMPACT analysis. *J Neurotrauma* 2007; 24:270-280.
26. McHugh GS, Engel DC, Butcher I, et al: Prognostic value of secondary insults in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:287-293.
27. McMahon CG, Yates DW, Campbell FM, et al: Unexpected contribution of moderate traumatic brain injury to death after major trauma. *J Trauma* 1999; 141:891-895.

28. Moskopp D, Staehle C, Wassmann H: Problems of the Glasgow Coma Scale with early intubated patients. *Neurosurg Rev* 1995; 18:253-257.
29. Miller JD, Sweet RC, Narayan R, et al: Early insults to the injured brain. *JAMA* 1978; 240:439-442.
30. Murray GD, Butcher I, McHugh GS, et al: Multivariate prognostic analysis in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:329-337.
31. Mushkudiani NA, Engel DC, Steyerberg EW, et al: Prognostic value of demographic characteristics in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:259-269.
32. Narayan RK, Greenberg RP, Miller JD, et al: Improved confidence of outcome prediction in severe head injury. A comparative analysis of the clinical examination, multimodality evoked potentials, CT scanning, and intracranial pressure. *J Neurosurg* 1981; 54:751-762.]

33. Perel P, Arango M, Clayton TMRC CRASH Trial Collaborators, et al: Predicting outcome after traumatic brain injury: practical prognostic models based on large cohort of international patients. *BMJ* 2008; 336:425-429.
34. Ramesh VG, Thirumaran KP, Raja MC. A new scale for prognostication in head injury. *J Clinical Neuroscience*. 2008; 15: 1110-1113.
35. Schreiber MA, Aoki N, Scott BG, et al: Determinants of mortality in patients with severe blunt head injury. *Arch Surg* 2002; 137:285-290.
36. Steyerberg EW, Mushkudiani N, Perel P, et al: Predicting outcome after traumatic brain injury: Development and international validation of prognostic scores based on admission characteristics. *PLoS Med* 2008; 5:e165.
37. Stocchetti N, Pagan F, Calappi E, et al: Inaccurate early assessment of neurological severity in head injury. *J Neurotrauma* 2004; 21:1131-1140.

38. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974; 2:81-84.
39. Van Beek JG, Mushkudiani NA, Steyerberg EW, et al: Prognostic value of admission laboratory parameters in traumatic brain injury: results from the IMPACT study. *J Neurotrauma* 2007; 24:315-328.
40. Wagner AK, Sasser HC, Hammond FM, et al: Intentional traumatic brain injury: epidemiology, risk factors, and associations with injury severity and mortality. *J Trauma* 2000; 49:404-410.
41. Walder AD, Yeoman PM, Turnbull A: The abbreviated injury scale as a predictor of outcome of severe head injury. *Intensive Care Med* 1995; 21:606-609.
42. Walia S, Sutcliffe AJ: The relationship between blood glucose, mean arterial pressure and outcome after severe head injury: an observational study. *Injury* 2002; 33:339-344.

PROFORMA

NAME :

I.P NO:

AGE:

D.O.A:

SEX:

D.O.D:

ADDRESS:

OCCUPATION:

LITERACY:

G.C.S ON ADMISSION:

G.C.S AFTER 24 HOURS:

OCULOCEPHALIC RESPONSE:

OCULOVESTIBULAR RESPONSE:

MODE OF INJURY:

PUPILLARY SIZE:

Rt

Lt

PUPILLARY REACTION TO LIGHT:

Rt

Lt

ADMISSION PULSE:

ADMISSION B.P:

RESPIRATORY RATE:

TEMPERATURE:

PRE EXISTING CO-MORBID ILLNESS:

BLOOD Hb%:

BLOOD UREA:

BLOOD SUGAR:

SERUM ELECTROLYTES:

BLOOD GROUPING:

BLEEDING TIME:

CLOTTING TIME:

CT SCAN FINDINGS:

MARSHAL CT CLASSIFICATION:

VENTILATORY CARE:

MANAGEMENT: CONSERVATIVE / SURGERY

TYPE OF SURGERY:

DURATION BETWEEN INJURY AND ADMISSION:

DURATION BETWEEN INJURY AND SURGERY:

POST OP COMPLICATIONS, IF ANY:

G.O.S:

ஆராய்ச்சி தகவல் அறிக்கை

தங்களது / தங்களது உறவனரது தலைக் காயம் பற்றிய ஆராய்ச்சி இது.

மதுரை அரசு இராஜாஜி பொது மருத்துவமனையில் தலைக்காயப் பிரிவில் சேரும் உள் நோயாளிகளின் தலைக்காயம் பற்றிய ஆராய்ச்சி நடைபெற்று வருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் தலைக்காயம் தொடர்பான காரணிகளும் அதன் முடிவுகளும் ஆராயப்பட்டு வருகின்றன. இந்த ஆராய்ச்சியில் அனைத்து தலைக்காய நோயாளிகளுக்கும் செய்யப்படும் உடற்கூறு செயல்பாடு பரிசோதனை, சி.டி.ஸ்கேன் மற்றும் இரத்தப் பரிசோதனைகளே இந்த ஆராய்ச்சியில் பங்கேற்போருக்கும் செய்யப்படுகின்றன. அதனால் தங்களுக்கு கூடுதல் பொருட் செலவோ அல்லது நோயின் ஆய்வறிக்கைக்கோ/ சிகிச்சைக்கோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சி முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

பங்கேற்பாளரின் கையொப்பம்

ஆராய்ச்சியாளரின் கையொப்பம்

நாள் :

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு :

பெயர் :

வயது :

பால் :

உள் நோயாளி எண் :

தலைக்காய சிகிச்சை எண் :

இந்த ஆராய்ச்சியின் விபரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கின்றேன்.

எனது / எனது உறவினரின் தலைக்காயம் தொடர்பான ஆராய்ச்சி இது என்பதை நான் நன்கு அறிவேன்.

எனது / எனது உறவினரின் தலைக் காயம் சம்மந்தமான காரணிகளும் அதன் முடிவுகளும் இந்த ஆராய்ச்சியில் ஆராயப்பட்டு வருகின்றன என்பதையும் நான் அறிவேன்.

இந்த ஆராய்ச்சியில் அனைத்து தலைக்காய நோயாளிகளுக்கும் செய்யப்படும் பரிசோதனைகளே எனக்கு/ எனது உறவினருக்கு செய்யப்படுகின்றன என்பதையும் நான் நன்கு அறிவேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி எனது சொந்த விருப்பத்தின் பெயரில்தான் பங்கு பெறுகின்றேன். மேலும் நான் இந்த ஆராய்ச்சியில் இருந்து எந்த நேரமும் பின் வாங்கலாம் என்பதையும்/ அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

(கையொப்பம்)

ABSTRACT

“ANALYTICAL STUDY OF PROGNOSTIC FACTORS AND OUTCOME IN TRAUMATIC BRAIN INJURY IN PATIENTS AGED 18 TO 40 YEARS”

Background: The incidence of head injury is increasing every year. All over the world, the majority of head injury victims of traffic accidents belong to the young and productive age group. Very often being the major earning members of the family the impact on young adults is many folds which cripples not only the individual but also the family and the nation.

Aim of the study: To study age as an important prognostic factor in the prediction of outcome in traumatic brain injury and to analyze the causes, other prognostic factors and outcome in traumatic brain injury in patients aged 18 to 40 years and to compare the same with other age groups.

Materials and methods: The patients belonging to the age group of 18 to 40 years admitted with traumatic brain injury in the Head Injury Ward, Government Rajaji Hospital, Madurai for a period of January 2010 to December 2011 were selected and the prognostic factors predicting the outcome of head injury in this age group were studied by comparing to two sets of control populations – one younger group (3 to 17 years) and another older group (>41 years) – admitted with traumatic brain injury in the same institution at the same period. In both study population and control groups, patients with polytrauma, alcohol intoxication, drug over dosage and patients with cerebro vascular accidents, spontaneous subarachnoid hemorrhage, patients in postictal state and patients with spinal cord injuries were excluded from the study. All the prognostic factors having significant impact over outcome in traumatic brain injury were recorded and analyzed on both univariate and multivariate analysis of individual factors with outcome.

Results and Conclusion: The study clearly showed that age is one of the strongest predictors of outcome after TBI. Among the study population the commonest cause of TBI is RTA, followed by falls and assaults. Among the various prognostic factors studied, age, motor score and pupillary reactivity bear a strong relationship with outcome as evidenced by univariate and multivariate analysis. The mild and moderate TBI according to Glasgow Coma Scale have a better prognosis than severe and critical TBI in all age groups. In the study group, patients with MHIPS of 15 - 18 had favorable outcome while those with <13 had unfavorable outcome. Bilateral absence of pupillary light reflex and impaired or absent oculocephalic response & oculovestibular response predicted a poor outcome. Diffuse injury IV of Marshall CT classification significantly correlated with poor outcome.

Key words: traumatic brain injury, age, glasgow coma scale, pupillary reaction, oculocephalic reflex, oculovestibular reflex, Marshall CT classification, Madras Head Injury Prognostic Score.

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
1	A	38	M	RTA	1	N	No	Yes	Yes	12.2	121	135	Normal	I	17	Alive		Good
2	A	22	M	Fall	2	AN	No	Yes	Yes	11.4	125	140	Normal	V	14		Alive	Good
3	C	62	M	RTA	4	AN	No	No	No	12.1	180	138	Normal	VI	8	Death		Death
4	A	34	M	RTA	2	AN	No	Yes	Yes	12.2	140	141	Normal	III	9		Death	Death
5	B	5	M	Assault	2	N	No	Yes	Yes	12.3	112	132	Normal	V	16		Alive	Good
6	C	49	M	Fall	3	AN	No	No	No	10.9	432	123	Normal	I	11	Death		Death
7	A	26	M	RTA	3	AN	No	No	Yes	10.8	133	122	Normal	IV	9		Death	Death
8	B	15	F	Assault	2	N	No	Yes	Yes	11.2	134	137	Normal	III	17		Alive	Good
9	A	19	M	RTA	1	N	No	Yes	Yes	12.1	134	138	Normal	I	17		Alive	Good
10	C	74	F	RTA	1	N	No	Yes	Yes	11.3	334	134	Abnormal	V	14		Alive	Good
11	B	17	F	RTA	1	N	No	Yes	Yes	12.6	113	134	Normal	V	16		Alive	Good
12	A	31	M	RTA	1	N	No	Yes	Yes	13.2	124	141	Normal	I	17	Alive		Good
13	A	40	F	Assault	3	N	No	Yes	Yes	11.2	136	138	Normal	II	13	Alive		Poor
14	A	27	M	RTA	2	AN	No	Yes	Yes	11.2	115	142	Normal	III	15		Alive	Good
15	B	12	M	Fall	1	N	No	Yes	Yes	11.3	122	138	Normal	I	18	Alive		Good
16	C	53	M	RTA	3	N	Yes	Yes	Yes	13.2	178	134	Normal	I	14	Alive		Poor
17	A	40	F	RTA	3	N	No	Yes	Yes	12.4	123	136	Normal	I	17	Alive		Good
18	C	69	M	RTA	4	AN	Yes	No	No	10.2	140	121	Normal	IV	8	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
19	B	16	M	RTA	4	AN	Yes	No	No	12.2	120	121	Abnormal	IV	10	Death		Death
20	A	38	M	Assault	4	AN	Yes	No	No	10.2	130	112	Abnormal	IV	9	Death		Death
21	A	23	M	Assault	3	AN	No	Yes	Yes	12.8	130	135	Normal	III	15		Alive	Good
22	B	14	M	RTA	2	N	No	Yes	Yes	12.6	113	134	Normal	I	18	Alive		Good
23	A	28	M	RTA	4	AN	Yes	No	No	11.8	126	129	Normal	I	11	Death		Death
24	C	48	M	Fall	4	AN	No	No	No	10.8	142	117	Normal	IV	8	Death		Death
25	C	63	F	RTA	3	AN	No	No	No	10.5	456	134	Normal	II	9	Death		Death
26	A	27	M	RTA	2	AN	No	Yes	Yes	11.4	135	143	Normal	IV	9		Death	Death
27	A	35	M	RTA	1	N	No	Yes	Yes	13.3	128	142	Normal	I	17	Alive		Good
28	A	38	M	RTA	3	AN	Yes	Yes	Yes	11.6	130	128	Abnormal	III	10		Death	Death
29	C	72	F	RTA	1	N	Yes	Yes	Yes	11.9	345	132	Abnormal	I	14	Death		Death
30	A	37	M	RTA	2	N	No	Yes	Yes	12.2	118	136	Normal	VI	17	Alive		Good
31	B	7	M	Fall	2	N	No	Yes	Yes	12.8	115	136	Normal	V	16		Alive	Good
32	A	22	M	RTA	3	AN	No	Yes	No	11.2	128	138	Normal	V	14		Alive	Good
33	A	24	M	RTA	1	N	No	Yes	Yes	13.7	124	140	Normal	I	17	Alive		Good
34	C	64	F	RTA	3	AN	No	No	No	10.8	342	125	Normal	IV	11	Death		Death
35	A	18	M	Fall	2	N	No	Yes	Yes	12.8	126	138	Normal	II	16	Alive		Good
36	B	15	M	RTA	3	AN	Yes	Yes	Yes	12.1	137	135	Abnormal	IV	10		Death	Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
37	A	36	F	RTA	1	N	Yes	Yes	Yes	14.1	121	145	Normal	I	17	Alive		Good
38	C	59	M	RTA	4	AN	No	Yes	Yes	11.7	163	128	Abnormal	IV	8	Death		Death
39	A	38	M	Assault	3	N	No	Yes	Yes	12.8	122	136	Normal	I	17	Alive		Good
40	C	66	M	RTA	1	N	No	Yes	Yes	11.2	128	135	Normal	II	15	Alive		Good
41	C	41	F	RTA	4	AN	No	Yes	Yes	12.6	130	138	Normal	V	13		Alive	Good
42	A	34	M	Assault	1	N	No	Yes	Yes	11.2	127	136	Normal	I	17	Alive		Good
43	A	27	M	RTA	2	AN	No	Yes	Yes	13.2	126	139	Normal	V	9		Death	Death
44	B	9	F	Fall	3	N	No	Yes	Yes	11.2	136	135	Normal	I	14	Alive		Poor
45	A	28	F	RTA	3	AN	Yes	Yes	Yes	12.4	136	136	Normal	III	15		Alive	Good
46	B	11	M	RTA	1	N	No	Yes	Yes	13.2	134	145	Normal	II	17	Alive		Good
47	A	37	M	RTA	2	AN	No	Yes	Yes	13.2	129	139	Normal	V	14		Alive	Good
48	C	68	F	Fall	2	AN	Yes	Yes	Yes	10.8	450	121	Abnormal	III	9		Alive	Poor
49	C	73	M	RTA	2	AN	No	Yes	Yes	12.3	333	134	Normal	V	13		Alive	Good
50	B	14	M	RTA	1	N	No	Yes	Yes	11.4	123	135	Normal	I	18	Alive		Good
51	A	19	M	Assault	2	N	No	Yes	Yes	10.8	135	140	Normal	I	17	Alive		Good
52	A	25	F	RTA	1	N	No	Yes	Yes	11.3	129	132	Normal	I	17		Alive	Good
53	B	12	M	Fall	1	N	No	Yes	Yes	11.8	134	145	Normal	II	17	Alive		Good
54	C	68	F	RTA	4	AN	No	Yes	Yes	11.5	420	123	Normal	IV	8		Death	Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
55	B	12	M	RTA	4	N	No	Yes	Yes	10.8	135	121	Normal	II	14	Alive		Poor
56	C	62	M	Fall	3	N	Yes	Yes	Yes	11.4	356	124	Normal	V	13		Alive	Good
57	A	26	M	RTA	4	N	No	Yes	Yes	12.6	122	136	Normal	IV	13		Alive	Good
58	A	33	M	RTA	2	N	No	Yes	Yes	13.5	112	143	Normal	I	17	Alive		Good
59	A	38	M	RTA	3	N	No	Yes	No	9.8	135	125	Normal	IV	12	Death		Death
60	C	74	M	RTA	3	AN	No	Yes	Yes	12.6	120	137	Abnormal	III	9		Alive	Poor
61	B	7	F	RTA	2	AN	No	Yes	Yes	11.2	138	121	Normal	V	11		Death	Death
62	A	19	M	RTA	2	AN	Yes	Yes	Yes	10.8	378	122	Abnormal	III	10		Death	Death
63	A	24	M	RTA	1	N	No	Yes	Yes	11.7	137	141	Normal	I	17	Alive		Good
64	B	13	M	RTA	1	N	No	Yes	Yes	11.9	118	137	Normal	IV	16		Alive	Good
65	C	71	M	Assault	4	N	No	Yes	Yes	12.1	138	135	Normal	II	13	Alive		Poor
66	A	28	M	RTA	2	AN	No	Yes	Yes	12.4	118	144	Abnormal	IV	14		Alive	Good
67	A	40	F	RTA	3	AN	Yes	Yes	Yes	12.6	450	131	Abnormal	V	15		Alive	Good
68	A	26	M	RTA	1	N	No	Yes	Yes	11.9	138	138	Normal	VI	17	Alive		Good
69	A	39	M	RTA	2	N	No	Yes	Yes	12.5	126	134	Normal	II	13	Alive		Poor
70	C	54	F	RTA	4	AN	No	No	No	11.3	277	127	Normal	IV	8	Death		Death
71	C	73	M	RTA	3	N	Yes	Yes	Yes	12.9	134	136	Normal	III	14		Alive	Good
72	B	17	M	RTA	3	N	No	Yes	Yes	12.2	128	112	Normal	I	14	Alive		Poor

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
73	C	62	M	Fall	2	N	No	Yes	Yes	12.8	132	135	Normal	I	16	Alive		Good
74	B	16	M	RTA	3	N	No	Yes	Yes	11.3	132	134	Normal	I	17		Alive	Good
75	A	29	F	Assault	2	N	Yes	Yes	Yes	11.5	119	136	Normal	II	17	Alive		Good
76	A	21	M	Fall	2	N	No	Yes	Yes	9.4	476	142	Normal	IV	12	Death		Death
77	A	31	F	RTA	3	N	No	Yes	Yes	11.8	110	137	Normal	I	14	Alive		Poor
78	A	36	M	RTA	3	AN	No	No	No	10.7	125	126	Normal	IV	9		Death	Death
79	A	33	F	Fall	4	AN	No	No	No	11.2	120	122	Abnormal	IV	9	Death		Death
80	C	66	M	RTA	4	AN	Yes	No	No	10.1	341	125	Abnormal	IV	8	Death		Death
81	B	10	F	RTA	3	N	No	No	Yes	10.8	126	121	Normal	VI	13	Death		Death
82	A	18	M	Assault	4	N	No	Yes	Yes	11.3	122	135	Abnormal	II	13	Alive		Poor
83	A	32	F	RTA	4	AN	Yes	Yes	Yes	12.8	135	135	Normal	V	9		Death	Death
84	A	22	M	RTA	2	AN	No	Yes	Yes	11.8	124	137	Normal	V	15		Alive	Good
85	C	41	M	RTA	2	N	No	Yes	Yes	12.4	134	132	Normal	V	14		Alive	Good
86	B	11	F	Fall	2	N	No	Yes	Yes	12.8	145	135	Normal	III	14	Alive		Poor
87	C	63	M	RTA	4	N	No	No	No	11.1	122	121	Normal	IV	12	Death		Death
88	A	37	M	RTA	4	AN	No	Yes	Yes	11.8	110	124	Normal	IV	9		Death	Death
89	A	27	M	RTA	1	N	No	Yes	Yes	12.9	139	135	Normal	I	17	Alive		Good
90	A	37	M	RTA	3	AN	No	No	Yes	11.6	135	140	Normal	III	14		Alive	Good

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
91	C	62	F	RTA	4	AN	No	Yes	Yes	12.4	141	120	Normal	VI	8	Death		Death
92	C	47	M	Assault	3	N	Yes	Yes	Yes	13.2	145	134	Normal	V	13		Alive	Good
93	A	21	M	RTA	2	N	No	Yes	Yes	12.6	125	139	Normal	II	16	Alive		Good
94	A	31	M	Fall	3	N	No	No	Yes	10.7	140	131	Normal	VI	12	Death		Death
95	B	13	M	RTA	3	N	No	Yes	Yes	12.4	118	118	Normal	II	14	Alive		Poor
96	A	39	F	RTA	3	N	No	Yes	Yes	13.6	116	138	Normal	I	17	Alive		Good
97	B	15	M	RTA	3	N	No	Yes	Yes	11.4	116	125	Normal	I	14	Alive		Poor
98	A	27	M	Fall	2	AN	No	Yes	Yes	13.2	128	140	Normal	III	14		Alive	Good
99	A	34	M	RTA	4	AN	Yes	No	No	10.8	122	132	Abnormal	VI	9	Death		Death
100	A	39	M	RTA	1	N	Yes	Yes	Yes	12.8	525	122	Normal	I	13	Death		Death
101	C	67	F	RTA	3	AN	Yes	Yes	Yes	13.1	121	135	Normal	V	8		Death	Death
102	A	27	M	Fall	3	AN	No	Yes	Yes	12.4	120	142	Normal	IV	9		Alive	Poor
103	A	34	M	RTA	2	AN	No	Yes	Yes	10.4	118	138	Normal	IV	9		Death	Death
104	C	84	F	Fall	2	N	No	Yes	Yes	12.3	132	137	Normal	VI	15	Alive		Good
105	B	14	M	RTA	1	N	No	Yes	Yes	12.7	124	135	Normal	II	17	Alive		Good
106	A	24	M	Fall	3	N	No	Yes	Yes	10.8	125	137	Normal	V	14		Alive	Good
107	A	33	M	Assault	3	AN	No	Yes	Yes	10.5	132	129	Normal	IV	9		Death	Death
108	B	12	F	RTA	4	AN	No	No	No	13.3	111	143	Normal	IV	10	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
109	C	73	M	Fall	3	AN	No	No	No	10.4	234	127	Normal	I	10	Death		Death
110	A	38	F	RTA	3	N	No	Yes	No	11.8	124	142	Normal	III	15		Alive	Good
111	A	22	M	Fall	2	AN	No	Yes	Yes	12.8	129	144	Normal	IV	13		Alive	Good
112	B	16	M	RTA	4	AN	No	Yes	Yes	12.8	130	136	Normal	V	15		Alive	Good
113	A	34	F	RTA	3	AN	No	Yes	Yes	12.2	128	139	Normal	IV	14		Alive	Good
114	A	21	M	Fall	4	AN	No	Yes	Yes	12	127	142	Normal	IV	14		Alive	Good
115	A	18	M	RTA	2	N	Yes	Yes	Yes	11.8	121	135	Normal	I	14	Alive		Poor
116	C	62	F	Fall	4	N	No	Yes	Yes	11.2	140	137	Normal	I	14	Alive		Poor
117	A	39	M	RTA	4	AN	No	No	No	10.7	121	118	Abnormal	IV	9	Death		Death
118	A	35	F	RTA	3	AN	No	No	Yes	11.6	126	141	Normal	III	14		Alive	Good
119	B	6	M	Fall	4	AN	Yes	No	No	11.3	122	115	Normal	IV	10	Death		Death
120	C	71	M	Assault	2	AN	YES	Yes	Yes	12.7	234	135	Normal	III	9		Death	Death
121	A	38	M	Fall	1	N	No	Yes	Yes	11.8	112	137	Normal	I	17	Alive		Good
122	C	58	F	RTA	1	N	No	Yes	Yes	12.5	124	138	Normal	I	16	Alive		Good
123	A	22	M	RTA	3	AN	No	No	Yes	11	135	123	Abnormal	V	10		Death	Death
124	B	13	F	Fall	2	N	No	Yes	Yes	12.5	123	138	Normal	V	15		Alive	Good
125	C	52	M	RTA	4	AN	No	No	Yes	12.8	256	129	Normal	V	13		Alive	Good
126	B	16	F	Fall	1	N	No	Yes	Yes	12.3	123	135	Normal	I	17	Alive		Good

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
127	A	31	M	RTA	1	N	No	Yes	Yes	10.8	108	132	Normal	I	17		Alive	Good
128	C	77	M	RTA	1	N	No	Yes	Yes	10.8	234	121	Abnormal	I	14	Death		Death
129	A	21	M	Fall	2	N	No	Yes	Yes	11.4	131	145	Normal	II	15	Alive		Good
130	A	25	M	Assault	4	AN	Yes	Yes	No	12.8	256	138	Normal	III	10		Death	Death
131	C	61	M	RTA	2	N	No	Yes	Yes	10.4	346	125	Abnormal	II	14	Alive		Poor
132	B	12	M	RTA	1	N	No	Yes	Yes	12.5	123	134	Normal	I	18	Alive		Good
133	A	27	M	Fall	3	N	No	Yes	Yes	13.2	96	149	Normal	I	14	Alive		Poor
134	A	36	F	RTA	1	N	No	Yes	Yes	12.1	107	138	Normal	I	17	Alive		Good
135	C	64	M	RTA	4	AN	Yes	No	No	12.6	121	135	Normal	V	13		Alive	Good
136	A	21	M	Fall	2	AN	No	Yes	Yes	11.6	120	131	Normal	IV	9		Death	Death
137	C	56	F	RTA	3	AN	Yes	Yes	Yes	11.8	412	127	Abnormal	IV	8		Death	Death
138	A	28	M	Assault	2	AN	No	Yes	Yes	11.5	134	141	Normal	V	14		Alive	Good
139	B	11	M	RTA	3	N	No	Yes	Yes	11.3	123	134	Normal	I	18		Alive	Good
140	A	37	M	RTA	4	N	Yes	Yes	Yes	12.5	116	122	Normal	VI	12	Alive		Poor
141	A	33	M	RTA	2	N	No	Yes	Yes	12.7	132	135	Normal	II	16	Alive		Good
142	A	31	M	Assault	3	N	No	Yes	Yes	13.2	107	135	Normal	I	17	Alive		Good
143	C	48	F	Fall	3	N	Yes	Yes	Yes	12.7	223	123	Normal	I	15	Alive		Poor
144	A	27	M	RTA	3	AN	Yes	Yes	No	11.2	136	126	Abnormal	IV	9	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
145	B	5	M	Fall	2	N	No	Yes	Yes	13.1	102	137	Normal	I	18	Alive		Good
146	C	43	M	RTA	1	N	No	Yes	Yes	12.5	145	139	Normal	III	16		Alive	Good
147	A	36	F	Fall	3	AN	No	No	Yes	11.8	78	139	Normal	II	11	Alive		Poor
148	A	24	M	RTA	2	AN	No	Yes	Yes	12.8	115	139	Normal	III	9		Death	Death
149	B	14	F	Fall	3	N	No	No	Yes	11.2	125	127	Normal	IV	12	Death		Death
150	A	29	F	RTA	4	AN	No	No	No	12.1	135	127	Normal	I	9	Death		Death
151	C	58	M	Assault	2	AN	No	Yes	Yes	13.2	324	137	Normal	IV	13		Alive	Good
152	A	19	M	RTA	3	AN	No	Yes	Yes	11.4	136	121	Normal	IV	9		Death	Death
153	C	72	M	Fall	2	N	No	Yes	Yes	12.3	138	130	Normal	II	15	Alive		Good
154	A	24	M	RTA	3	N	Yes	Yes	Yes	12.8	118	141	Normal	II	14	Alive		Poor
155	B	16	M	RTA	2	N	No	Yes	Yes	11.3	129	139	Normal	V	15		Alive	Good
156	A	35	M	Fall	4	AN	No	No	No	10.5	124	120	Abnormal	IV	10	Death		Death
157	A	28	M	RTA	2	AN	No	Yes	Yes	13.3	136	132	Normal	III	15		Alive	Good
158	C	72	F	RTA	4	AN	No	Yes	Yes	12.8	386	132	Normal	VI	8		Death	Death
159	A	24	M	Fall	3	AN	No	Yes	Yes	12.2	136	145	Normal	V	14		Alive	Good
160	B	14	M	RTA	1	AN	No	Yes	Yes	11.4	134	144	Normal	III	16		Alive	Good
161	A	31	F	RTA	2	N	No	Yes	Yes	11.4	135	133	Normal	I	13	Alive		Poor
162	C	65	M	Fall	2	AN	No	Yes	Yes	12.7	335	138	Normal	V	13		Alive	Good

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
163	A	22	F	RTA	1	N	No	Yes	Yes	11.3	106	133	Normal	I	17	Alive		Good
164	C	49	M	RTA	4	AN	No	No	Yes	11.8	321	124	Normal	IV	8	Death		Death
165	A	31	M	Assault	2	N	No	Yes	Yes	12.6	119	136	Normal	II	16	Alive		Good
166	B	6	M	RTA	2	N	No	Yes	Yes	12.7	112	137	Normal	III	16		Alive	Good
167	A	37	M	Fall	2	AN	No	Yes	Yes	11.3	121	138	Normal	IV	14		Alive	Good
168	C	63	F	RTA	2	N	No	Yes	Yes	12.5	128	137	Normal	II	15	Alive		Good
169	A	40	F	RTA	4	AN	No	No	No	10.8	128	125	Normal	IV	9	Death		Death
170	A	21	M	Fall	3	AN	Yes	Yes	No	12.4	124	125	Abnormal	IV	9		Death	Death
171	C	76	M	RTA	3	AN	Yes	No	No	10.8	276	128	Normal	VI	8	Death		Death
172	A	36	M	RTA	2	N	Yes	Yes	Yes	13.4	312	143	Normal	V	11	Death		Death
173	A	20	M	Fall	3	N	No	Yes	Yes	12.5	108	136	Normal	II	14	Alive		Poor
174	C	63	M	Assault	4	AN	Yes	No	No	11.2	417	128	Normal	V	13		Alive	Good
175	A	18	F	RTA	4	AN	No	Yes	Yes	11.8	135	143	Normal	V	14		Alive	Good
176	A	36	M	RTA	1	N	No	Yes	Yes	10.2	497	131	Abnormal	I	13	Death		Death
177	B	13	M	Fall	2	N	No	Yes	Yes	13.2	103	138	Normal	VI	18	Alive		Good
178	A	31	M	RTA	2	N	No	Yes	Yes	12.8	121	135	Normal	II	12	Alive		Poor
179	A	29	M	Fall	4	AN	Yes	Yes	Yes	12.5	121	138	Normal	V	9		Death	Death
180	C	68	F	RTA	2	AN	No	No	No	12.3	234	139	Abnormal	I	13	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
181	B	3	M	RTA	4	AN	No	No	No	10.4	127	128	Normal	IV	15		Alive	Good
182	C	55	F	Assault	1	N	No	Yes	Yes	12.7	135	130	Normal	I	16	Alive		Good
183	A	30	M	Fall	3	AN	No	No	No	12.8	412	145	Abnormal	V	9		Death	Death
184	A	18	M	RTA	4	AN	Yes	No	No	11.3	134	117	Abnormal	IV	9	Death		Death
185	C	74	F	Assault	2	AN	Yes	Yes	Yes	11.9	325	118	Abnormal	V	8		Alive	Poor
186	A	26	M	RTA	3	AN	No	Yes	Yes	12.6	120	127	Normal	IV	9		Death	Death
187	C	69	F	Fall	3	AN	No	Yes	Yes	12.8	126	137	Normal	III	9		Death	Death
188	A	29	M	RTA	4	AN	No	No	No	11.5	136	127	Abnormal	IV	9	Death		Death
189	A	25	M	RTA	2	N	No	Yes	Yes	11.4	127	139	Normal	II	15	Alive		Good
190	B	11	M	Fall	2	N	No	Yes	Yes	12.3	145	139	Normal	V	15		Alive	Good
191	A	21	F	Fall	3	N	No	Yes	Yes	11.5	104	137	Normal	I	14	Alive		Poor
192	C	73	M	RTA	1	AN	No	Yes	Yes	11.7	356	123	Normal	V	13		Alive	Good
193	C	81	M	Assault	3	AN	No	No	Yes	11.5	245	132	Normal	II	10	Death		Death
194	A	19	M	RTA	3	AN	No	Yes	Yes	11.5	138	140	Normal	III	15		Alive	Good
195	B	16	M	RTA	2	N	No	Yes	Yes	13.1	129	132	Normal	V	15		Alive	Good
196	A	35	F	RTA	3	AN	No	No	Yes	12.8	128	129	Normal	IV	10		Death	Death
197	A	28	M	RTA	4	N	No	Yes	Yes	11.7	114	134	Normal	II	13	Alive		Poor
198	A	39	M	Fall	2	AN	No	Yes	Yes	12.3	124	137	Abnormal	V	15		Alive	Good

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
199	C	58	M	RTA	2	AN	No	Yes	Yes	12.4	123	137	Normal	IV	8		Death	Death
200	A	25	M	RTA	3	N	No	Yes	Yes	12.4	100	142	Normal	I	17	Alive		Good
201	B	17	F	Fall	3	N	No	Yes	Yes	11.8	110	122	Normal	III	14	Alive		Poor
202	A	31	M	Assault	3	AN	No	Yes	No	12.3	126	143	Normal	IV	9		Alive	Poor
203	C	80	F	RTA	4	AN	No	No	No	13.6	256	126	Abnormal	IV	8	Death		Death
204	A	29	M	RTA	2	N	No	Yes	Yes	12.9	122	131	Normal	V	14		Alive	Good
205	C	46	M	Fall	2	N	No	Yes	Yes	13.2	136	140	Normal	I	16	Alive		Good
206	A	34	M	RTA	1	N	No	Yes	Yes	10.7	98	137	Normal	I	17	Alive		Good
207	A	27	M	RTA	3	N	No	No	No	10.2	137	121	Normal	IV	12	Death		Death
208	B	14	M	Fall	1	N	No	Yes	Yes	11.6	126	145	Normal	V	16		Alive	Good
209	A	19	M	RTA	4	AN	No	Yes	Yes	10.8	138	129	Normal	IV	9		Death	Death
210	C	57	M	RTA	3	AN	Yes	Yes	Yes	12.6	256	123	Normal	IV	8		Death	Death
211	A	26	F	Fall	4	AN	No	No	Yes	11.4	140	132	Normal	III	10		Death	Death
212	A	33	M	RTA	2	AN	No	Yes	Yes	12.6	125	145	Normal	V	9		Death	Death
213	B	15	M	Assault	1	N	No	Yes	Yes	11.4	134	134	Normal	II	17	Alive		Good
214	C	62	M	RTA	1	N	No	Yes	Yes	10.2	341	126	Normal	I	14	Death		Death
215	A	27	F	RTA	3	N	No	Yes	Yes	12.6	131	136	Normal	III	16		Alive	Good
216	A	19	M	RTA	4	AN	Yes	Yes	Yes	12.2	136	137	Abnormal	IV	14		Alive	Good

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
217	A	27	M	RTA	4	N	Yes	No	Yes	13.6	137	118	Normal	VI	12	Death		Death
218	C	69	M	Fall	3	AN	No	Yes	No	11.7	341	129	Normal	I	10	Death		Death
219	A	20	M	RTA	2	N	No	Yes	Yes	11.7	120	137	Normal	II	16	Alive		Good
220	B	13	F	RTA	1	N	No	Yes	Yes	12.4	134	143	Normal	I	18	Alive		Good
221	A	34	M	RTA	4	AN	Yes	No	No	10.8	128	119	Normal	VI	10	Death		Death
222	C	53	M	RTA	4	AN	No	No	No	11.6	312	127	Normal	VI	8	Death		Death
223	A	31	M	Assault	2	N	No	Yes	Yes	12.2	128	135	Normal	I	13	Alive		Poor
224	A	33	M	Fall	1	N	Yes	Yes	Yes	12.7	118	139	Normal	I	17	Alive		Good
225	C	74	M	Fall	2	N	No	Yes	Yes	12.7	129	129	Normal	V	14		Alive	Good
226	A	27	F	Fall	4	AN	No	No	No	10.6	139	130	Abnormal	IV	9	Death		Death
227	A	38	M	RTA	4	N	No	Yes	Yes	10.7	110	136	Normal	I	12	Alive		Poor
228	C	67	M	Fall	3	N	Yes	No	No	12.4	321	121	Normal	IV	13	Death		Death
229	B	15	M	Fall	3	N	No	Yes	Yes	12.3	123	134	Normal	II	17		Alive	Good
230	A	31	F	RTA	3	AN	No	Yes	No	12	136	121	Abnormal	III	10		Death	Death
231	A	38	M	RTA	2	N	No	Yes	Yes	13.5	130	143	Normal	II	17	Alive		Good
232	C	78	M	Fall	3	AN	No	No	No	12.9	234	120	Normal	I	10	Death		Death
233	A	27	M	RTA	4	AN	No	Yes	Yes	12.2	136	128	Abnormal	III	11		Death	Death
234	B	14	M	Fall	2	AN	No	Yes	Yes	11.7	140	118	Normal	IV	11		Death	Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
235	C	51	M	Assault	3	AN	No	Yes	Yes	12.7	134	132	Normal	III	14		Alive	Good
236	B	12	M	RTA	3	N	No	Yes	Yes	12.7	134	137	Normal	I	18		Alive	Good
237	A	37	M	RTA	3	N	No	Yes	Yes	11.2	122	142	Normal	II	12	Alive		Poor
238	A	26	M	RTA	4	AN	Yes	No	No	11.8	140	134	Abnormal	IV	9	Death		Death
239	C	62	F	RTA	2	AN	Yes	Yes	Yes	11.4	156	130	Normal	V	8		Death	Death
240	C	77	M	Fall	2	AN	No	Yes	Yes	12.8	124	128	Normal	V	13		Alive	Good
241	A	30	M	RTA	2	N	No	Yes	Yes	12.7	111	141	Normal	II	16	Alive		Good
242	Z	16	M	RTA	1	N	No	Yes	Yes	12.5	134	137	Normal	I	18	Alive		Good
243	A	27	M	Assault	1	N	No	Yes	Yes	12.8	125	143	Normal	I	17	Alive		Good
244	C	68	M	RTA	3	AN	No	Yes	Yes	12.1	134	138	Normal	IV	13		Alive	Good
245	A	24	M	Fall	3	N	Yes	Yes	Yes	13	132	129	Normal	IV	11		Death	Death
246	C	64	M	RTA	2	N	No	Yes	Yes	13.4	145	146	Normal	II	15	Alive		Good
247	A	28	F	Fall	2	AN	No	Yes	Yes	12	136	138	Normal	IV	9		Death	Death
248	B	12	F	RTA	2	N	No	Yes	Yes	12.8	127	140	Normal	V	16		Alive	Good
249	C	59	M	Fall	2	N	Yes	No	No	13.2	187	137	Abnormal	II	14	Death		Death
250	A	39	M	RTA	4	AN	No	Yes	Yes	12.6	127	122	Normal	V	9		Death	Death
251	C	48	M	RTA	2	N	No	Yes	Yes	12.1	345	119	Abnormal	I	14	Alive		Poor

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
252	B	4	M	Fall	1	AN	No	Yes	Yes	13.2	123	145	Normal	III	14		Alive	Good
253	C	57	M	Assault	2	AN	Yes	Yes	Yes	12.3	145	137	Normal	V	8		Death	Death
254	A	28	F	RTA	1	N	No	Yes	Yes	12.6	122	145	Normal	I	17	Alive		Good
255	A	35	M	RTA	2	N	No	Yes	Yes	12.4	116	140	Normal	I	17	Alive		Good
256	A	31	M	RTA	4	AN	No	Yes	Yes	12.4	126	139	Normal	V	15		Alive	Good
257	C	68	M	RTA	3	AN	No	No	No	12.6	245	127	Normal	I	10	Death		Death
258	B	15	F	RTA	3	AN	No	Yes	Yes	12.7	118	129	Normal	IV	10		Death	Death
259	A	21	M	Fall	3	AN	No	Yes	Yes	11.4	133	138	Normal	V	15		Alive	Good
260	A	40	M	RTA	2	N	No	Yes	Yes	12	118	138	Normal	II	16	Alive		Good
261	C	47	M	RTA	2	AN	No	No	No	12.5	206	132	Abnormal	I	13	Death		Death
262	B	15	M	Fall	2	N	No	Yes	Yes	12.6	124	139	Normal	VI	18	Alive		Good
263	C	49	M	RTA	4	N	No	Yes	Yes	11.7	127	139	Normal	I	13	Alive		Poor
264	A	38	F	Assault	3	AN	Yes	No	Yes	11.8	375	128	Normal	III	14		Alive	Good
265	A	31	M	RTA	4	AN	No	No	No	11.9	141	124	Abnormal	IV	9	Death		Death
266	C	61	M	Fall	3	AN	No	Yes	Yes	11.7	238	121	Abnormal	V	8		Death	Death
267	B	10	M	RTA	1	N	No	Yes	Yes	11.3	123	142	Normal	IV	16		Alive	Good
268	A	25	M	RTA	2	AN	No	Yes	Yes	11.6	128	136	Normal	V	15		Alive	Good
269	C	78	M	Fall	2	N	Yes	No	No	12.6	245	138	Abnormal	I	15	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
270	A	36	M	RTA	3	N	No	Yes	No	11.8	148	138	Normal	VI	11	Death		Death
271	A	31	M	Assault	2	N	No	Yes	Yes	11	116	132	Normal	I	13	Alive		Poor
272	B	13	M	Fall	4	N	No	Yes	Yes	11.8	118	131	Normal	I	14	Alive		Poor
273	A	40	M	RTA	4	N	No	Yes	Yes	11.9	128	130	Normal	I	12	Alive		Poor
274	C	46	M	RTA	4	AN	No	Yes	Yes	12.8	120	136	Normal	III	14		Alive	Good
275	A	31	M	RTA	3	N	No	Yes	Yes	13.2	112	129	Normal	II	14	Alive		Poor
276	A	27	F	RTA	2	N	No	Yes	Yes	10.9	125	135	Abnormal	II	17	Alive		Good
277	C	59	F	Fall	1	N	No	Yes	Yes	14	128	145	Normal	I	16	Alive		Good
278	A	33	M	Fall	4	AN	No	No	No	12.8	131	121	Abnormal	IV	9	Death		Death
279	A	28	M	RTA	3	N	No	Yes	Yes	13	118	140	Normal	I	17	Alive		Good
280	A	31	M	RTA	4	AN	No	No	No	11.8	135	130	Normal	III	11		Death	Death
281	C	43	M	Assault	3	N	Yes	Yes	Yes	12.5	178	137	Normal	I	16	Alive		Good
282	A	36	M	Fall	3	AN	No	No	Yes	10.5	135	142	Normal	III	14		Alive	Good
283	A	36	M	RTA	3	AN	No	No	Yes	12.6	130	128	Normal	IV	9		Death	Death
284	A	21	F	RTA	2	N	No	Yes	Yes	11.7	113	138	Normal	II	16	Alive		Good
285	C	48	M	Assault	2	N	Yes	Yes	Yes	12.3	289	120	Normal	II	14	Alive		Poor
286	A	38	M	RTA	3	AN	No	Yes	Yes	11.8	125	143	Normal	III	14		Alive	Good
287	C	77	M	Fall	3	AN	No	No	No	13.2	256	121	Normal	I	10	Death		Death

Sl.No	Study Group	Age (years)	Sex	Injury	GCS (1=14&15, 2=9-13, 3=5-8, 4=3&4)	Pupils (N= Normal, AN= Abnormal)	Hypotension	OCR	OVR	Hb (gm %)	Glucose(mg %)	Sodium (mEq/L)	Coagulation profile (BT, CT)	Marshall CT classification	MHIPS Score	Conservative	Surgery	GOS
288	B	4	F	Fall	2	N	No	Yes	Yes	12.3	116	140	Normal	I	17	Alive		Good
289	A	21	M	RTA	4	AN	Yes	No	No	12.5	121	130	Normal	IV	9	Death		Death
290	C	64	M	Fall	2	N	Yes	Yes	Yes	12.7	256	140	Normal	IV	13		Alive	Good
291	A	26	M	RTA	3	N	No	Yes	Yes	13.5	122	143	Normal	I	17	Alive		Good
292	C	69	F	Fall	4	AN	Yes	No	No	11.8	240	121	Normal	IV	13		Alive	Good
293	A	39	M	Assault	1	N	No	Yes	Yes	12.4	127	135	Normal	I	17		Alive	Good
294	A	34	M	Fall	3	N	No	No	Yes	12.3	120	139	Abnormal	IV	13	Death		Death
295	B	15	F	Fall	1	N	No	Yes	Yes	11.5	134	145	Normal	II	15	Alive		Good
296	C	57	M	RTA	2	N	No	Yes	Yes	12.8	129	141	Normal	VI	15	Alive		Good
297	A	20	M	Fall	4	AN	No	No	No	12.7	120	126	Abnormal	IV	9	Death		Death
298	A	32	F	RTA	4	N	No	No	No	11.2	126	127	Abnormal	VI	11	Death		Death
299	C	71	M	Fall	3	N	Yes	No	NO	11.7	278	120	Normal	VI	13	Death		Death
300	A	27	M	Fall	2	N	No	Yes	Yes	12.4	127	139	Normal	II	16	Alive		Good